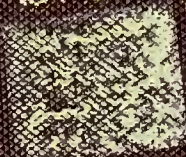


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PERIODONTAL DISEASE

AND ITS

TREATMENT BY IONIC MEDICATION

BY

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PREFACE.

THIS book, which is based on conclusions reached after over thirty years of experience and study of this most difficult problem, is intended to emphasize the local aspect of periodontal disease and to demonstrate that much can be done in combating the disease by systematizing a definite method of treatment to the exclusion of conflicting opinions of many writers on vital points of the subject.

In adopting the view that periodontal disease is purely local *in origin*, the writer has gone to much pains to draw attention to the incipient stages of the disease and to point out that those who ignore or overlook the importance of this stage do injustice to their patients.

Pyorrhœa alveolaris, it has been shown, is but a termination of periodontal disease which can be averted by a systematized method of dealing with the premonitory symptoms, but these are so often overlooked that the disease is now almost universal in the mouths of adult people, even in those who frequently consult dentists.

A strong protest has been entered against reckless wholesale extraction of teeth as the only means of curing the disease; many practitioners seem to overlook the symptoms of approaching pyorrhœa, or entertain pessimistic views on the arrest of the disease by local measures and eventually resort to extraction of every tooth in the denture; this is but

admission of failure to cope with scientific problems which it should be our duty to undertake.

The extraction of certain teeth for the benefit of the patient and in the interest of treatment of other teeth is undoubtedly often necessary but a high standard should be placed on the importance of retaining the organs of mastication and the elimination of oral sepsis by available methods.

Treatment by ionic medication which is advocated, is intended to cope with one etiological factor but stress is laid on the futility of depending on it alone and the importance of dealing with many other factors which are specified.

It is hoped that some useful hints are given by classifying the stages of the disease and giving a definite method of treatment for each, also, subdividing the pyorrhœa stage with a view of drawing attention to the different phases which present knotty problems in the treatment of the disease.

No claim is made that this is the *only* method of treating pyorrhœa alveolaris; on the contrary, the writer is aware that many are curing the disease by highly scientific methods which, if examined, in every case will be found to consist of a definite system followed on lines of strictest attention to *local* etiological factors.

A short chapter is given on the principle of ionic medication, the apparatus and the technic necessary to carry out this method of applying drugs; the scope of this work does not admit of details on electro-therapeutics or electrophysics but it is advisable that a thorough knowledge of these should be acquired by those who adopt this treatment, and the writer advises them to consult larger works on the subject both from a medical and dental standpoint.

I desire to take this means of expressing my appreciation

of the valuable assistance rendered by friends in the preparation of this work, particularly Dr. E. D. Barrows and Dr. N. S. Finzi, who kindly provided some of the *x*-ray illustrations; to Dr. E. C. Kirk, for advice on the general context, and Messrs. Ash & Son and De Tray & Co., for loan of blocks.

E. S.

LONDON, 1919.

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PERIODONTAL DISEASE.

INTRODUCTION.

It would be difficult to name a disease associated with more controversial literature than "pyorrhœa alveolaris." Historical evidence places it in the category of one of the oldest existing maladies, for anatomical specimens in museums which date back to many centuries B.C. reveal unmistakable evidence of the disease as known to us today. Writers about the middle of the eighteenth century first began to describe it, and later on, in the early part of the nineteenth century, more frequent reference was made to this disorder. About the year 1875 Dr. Riggs first accurately described the disease; he regarded it as purely local, and treated it from that aspect in a highly satisfactory manner. Since then the etiology, pathology and treatment of periodontal disease has attracted ever-increasing interest and been debated on such controversial lines that it has become well-nigh impossible to determine, without vast personal experience, which are the correct deductions to accept. The medical profession has recently discovered the disease, and are now imposing their will on the dental profession; some are treating it themselves by vaccines, others are condemning all teeth affected with the disease to the forceps, while others are content with attributing nearly every disease of the

human body to the effects of oral sepsis and saddling the dentist with the responsibility of treatment.

But dentists themselves have not settled on any fixed etiology or pathology, and consequently vary in lines of treatment to a remarkable degree, and a serious feature of consequent inability to cope with the disease is a strong conviction prevalent that under no circumstances is it curable when the teeth are retained. There is usually a vague or controversial element introduced by even the most recent writers on the subject, making it difficult for the most ardent student to determine what is meant or which conclusions are the correct ones. The disease is usually studied in the advanced stages, the preliminary stages being overlooked as a gingival affection with no direct bearing.

Howe¹ in his report on pathology and etiology to the sixth International Dental Congress states: "This fully developed pathological condition presents three distinct phases. . . . I refer to bacterial activity, pathological calcifications, tissue degeneration." The last of these is a vague statement over which great division of opinion exists and refers to advanced stage.

In a recent and important work—*The Science and Practice of Dental Surgery*, by Norman Bennett, is recorded: "The causes of chronic suppurative periodontitis are at present very vaguely understood, some writers attribute the disease to *Constitutional Disorders*, while others consider it to be due to local irritants. The consensus of opinion seems to favor the belief that both play an important part."

A great deal of stress is laid on the constitutional causes by the vast majority of writers, but local causes are always

¹ Sixth Inter. Dent. Congress, p. 115.

included and so mixed up as to make the student wonder which is really the important factor.

Talbot presented a mass of evidence to demonstrate "that the causes of interstitial gingivitis are divisible into predisposing causes (which may be subdivided into local predisposing and constitutional) and exciting causes. The exciting causes are either constitutional or local, but as a rule are local or have local action."

For many years the disease was associated with gout, and uric acid was made responsible for at least one form of pyorrhœa,¹ "in which the local necrosis of the periodontal membrane is caused by gouty disease of one of the blood-vessels in its substance."

Newland Pedley² concluded that "pyorrhœa alveolaris is essentially of constitutional origin," and asserts that "the weight of evidence tends to place pyorrhœa alveolaris in the category of bone disease."

Hopewell-Smith,³ in his research on Pathohistology of the disease, considers that "pyorrhœa alveolaris does not commence as a gingivitis," but that "it is essentially dependent upon an osseous lesion."

H. P. Pickerill⁴ asserts that "The direct cause in all cases is infection by pathogenic organisms."

Miller⁵ says: "It is still a matter of debate whether a local irritant be at all required to the origination of the disease. As regards the participation of bacteria in pyorrhœa alveolaris, our present knowledge of suppurative inflammations compels us to consider the former as the cause of the suppura-

¹ Fitzgerald: Pyorrhœa Alveolaris.

² Dental Record, May, 1887.

³ Pyorrhœa Alveolaris: Its Pathohistology, Dental Cosmos, April, 1911, p. 397.

⁴ Stomatology in General Practice, p. 11.

⁵ The Microorganisms of the Human Mouth, p. 332.

tions incident to the disease. Microörganisms which possess pyogenic properties, temporarily or permanently, inhabit every mouth. If, therefore, the power of resistance of the periodontal membrane be impaired by any one of the above-mentioned local or constitutional causes in such a manner as to furnish a suitable culture medium for the bacteria, they will, of course, begin their ravages and the usual symptoms will follow."

G. V. Black was the first to classify the disease from symptoms observed into two classes, both closely allied "gingivitis" and "phagedenic pericementitis," the former dependent on the presence of two forms of calculus and the latter, of a more destructive character, he associated with a specific form of microörganism, at the time undiscovered, as well as the presence of calculus, which causes "an inflammation of a peculiar character which results in a destruction of the periodontal membrane."¹ Black's conception of the disease at that time holds good now in many respects. Knowledge has been added, but his teachings on the subject laid the foundation for much of the conservative work now done.

Recent investigations on the protozoa of the buccal cavity by A. Chiavaro,² of Rome, and M. T. Barrett,³ of Philadelphia revealed the presence of *Entamœbæ buccalis* almost constantly in pyorrhœa pus, and Barrett leans toward the view that this protozoa causes the disease, while Chiavaro holds the opposite view.

Colyer,⁴ studying the anatomy of chronic general periodontitis, draws the conclusion "(1) that the bone lesion is a progressive rarefying osteitis commencing at the margin

¹ Black, G. V.: *Am. System of Dentistry*, i, 954.

² *Dental Cosmos*, September, 1914, p. 1089.

³ *Ibid.*, December, 1914, p. 1345.

⁴ *Chronic General Periodontitis*, pp. 30 and 64.

of the alveolar process, and (2) that the varying density of the bone influences the rate of destruction." The cause of the disease in man he attributes "to the disease being started by injury of the gingival margin from food débris or the local action of toxins as seen on the marginal gingivitis of mouth breathers." He also points out that "The prevalence of the disease is probably due to the character of the diet of the present day."

Znamensky,¹ studying the disease from the anatomopathological standpoint, concluded that constitutional disorders as well as local irritants are responsible for osteoporosis characteristic of the disease. He says: "The different diseases which produce an osteoporosis of the bones in the whole system, and particularly in the sockets, may be the predisposing cause of alveolar pyorrhœa. The diseases of general constitutional character which belong to this class are osteomalacia, rickets, scrofula, syphilis, acute eruptive processes, fevers and typhus. Further, there are also diseases of the blood, exchange of matter in the system, such as anemia, chlorosis, scurvy, leukæmia, hemophilia, diabetes mellitus and gout. The causes of atrophy of the tissues are scanty nourishment, repeated pregnancy, chronic catarrh of the stomach, diseases which exhaust the system, tabes dorsalis, tuberculosis and rheumatism." Referring to the local causes, on which he lays very little stress, he says: "The most frequent of these is an accumulation of tartar deposits." He also vaguely refers to local influence of irregularities of teeth and the use of soft foods.

These divergent views on etiology and pathology of periodontal disease here mentioned are but a few selected from

¹ XVIIth International Congress of Medicine, Section XVII, p. 26.

some of the best known recent writers—many more might be quoted to emphasize the confusion which exists regarding the character of the disease. The great lack of some recognized system in the treatment of the disease is undoubtedly due to these divergencies of opinion as to its cause, for all treatment is based on the conception of certain etiology—those who believe in constitutional causes, or local causes or gouty diathesis, or osseous lesion or pathogenic organisms, or protozoa, base their treatment accordingly, and unfortunately the majority have little real conviction that any form of treatment is likely to succeed.

CHAPTER I.

ETIOLOGY OF PERIODONTAL DISEASE.

No useful etiology of periodontal disease can be based on the conception that pus must be visible or present in the diagnosis of the disease. When the recognition of the disease is delayed until this stage is reached, the most important stages have simply been overlooked until the disease has been in existence for a very long time, and it is on this account that the very term pyorrhœa, employed to express it, is bad and misleading. When pus is exuding or can be expressed from the sides of the teeth, the disease has already advanced considerably.

A study of the pathology of the disease should leave no doubt in our minds that it is of purely local origin; it only needs to be recognized in the incipient stages to impress us with the force of this conclusion. The vast majority of cases have some local irritant cause or series of local irritant causes directly responsible for the starting of that inflammation which lends itself to the possibility of septic infection of the adjacent tissues.

The incipient stage of the disease is to be found about the gingival margin in what is usually termed gingivitis and not generally associated with pyorrhœa. At this stage if the gingival trough (Fig. 1) or space be examined, it will be found to contain some local irritant in the form of salivary calculus or stagnant food or coating of translucent mucous

extending from the necks of the teeth into the trough, or, in the case of badly kept teeth, quantities of salivary calculus on the teeth impinging on the gingival margin. This local irritant causes a slight inflammation of the gingival margin, which may be general or may be confined to the dental papillæ, but wherever there is foreign matter in contact with the delicate epithelial lining of that fold of gum which constitutes one boundary of the gingival trough, inflammation occurs. This inflammation may not be visible on the outer surface of the gum (as it often is not in well kept

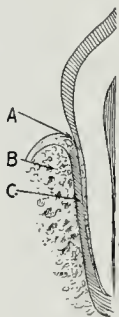


FIG. 1.—A, gingival trough; B, alveolus; C, periodontal membrane.

teeth which are constantly massaged by the brush) but may be confined to the gingival trough, extending chiefly in the direction of the upper fibers of the periodontal membrane. Mouth bacteria are ever present, and when a stagnation area is produced it is impossible to designate the precise stage at which proliferation of pathogenic bacteria first takes place; it is probably at a much earlier stage than usually estimated.

This local inflammation, if left unchecked for any considerable length of time, or if allowed to recur at frequent intervals,

leads to the next stage in the disease, in which the local irritant is augmented by one of the products of inflammation, which is subgingival or *serumal calculus*. Considerable doubt has been expressed by some authors on the existence of this form of calculus, and many writers ignore it altogether, but there is nothing so certain as the production of this form of deposit, when inflammation of the gingival fold occurs.

Subgingival Calculus.—Subgingival calculus is formed at a very early stage in the gingival inflammation, which precedes chronic periodontal disease, and is one of the primary causes of the severing of the fibers of the periodontal membrane, and the rarefying osteitis associated with pyorrhœa alveolaris.

This form of calculus is derived from “an exudation of mucus rich in colloidal material,”¹ which exudes from the inflamed surface of the gingival fold in direct contact with the necks of the teeth; it is a product of the blood, consisting of inorganic salts, principally magnesium phosphate, calcium phosphate and calcium carbonate; the predominating salt being dependent upon the amount of surcharge of that particular salt present in the blood of the individual. The amount of subgingival calculus derived from so small an area of inflammation is no doubt exceedingly small at first, and in the early stages of its formation on the roots of the teeth only amounts to a delicate granular layer, which is difficult to detect, but gradually the deposit increases, and itself becomes a source of further irritation to the inflamed area immediately covering it. It further increases, until it is readily discernible in the form of granular nodules or a hard, smooth, brownish crust, which adheres firmly to the necks of the teeth.

Subgingival calculus having once been deposited, inflamma-

¹ Kirk: Operative Dentistry, p. 484.

tion seems to take on a more acute form, the gums become turgid and heaped up in the interdental spaces (increasing the depth of the gingival trough), and become loosened from the necks of the teeth, forming a suitable receptacle for particles of food. The inflammation extends to the periodontal membrane and the alveolar bone, the former becomes detached and recedes, forming a small pocket, the latter is absorbed gradually from its free margin toward the body of the bone, and a process of rarefying osteitis starts (Fig. 2). At this stage bacteria undoubtedly exercise a great



FIG. 2.—Starting rarefying osteitis.

influence—hastening the progress of the disease. The contents of the gingival trough now consist of desquamation of the epithelial lining of the gingival trough, dead tissue cells, leucocytes and serum exuding from the inflamed surface, mixed with mucus and particles of decomposing food, altogether providing a most admirable culture medium which is kept at the right temperature for the development and maintenance of pathogenic organisms.

Up to this stage no pus is visible and pressure on the gums only produces bleeding, but the soft tissues are in a diseased state and bacteria contained in the gingival trough find

ready access into the intercellular spaces and capillaries, and probably penetrate into the alveolar bone.

In many well kept mouths some of these symptoms are entirely absent—there is no swelling or turgidity of the gums, no discomfort, the gingival trough, instead of being widened for the reception of foreign matter, appears to be shrinking gradually. The gingival margin is thinned and taut about the necks of the teeth, the alveolar border has obviously absorbed away, and the dental papillæ have disappeared leaving the necks of the teeth well defined, revealing wide interspaces. The teeth may be beautifully kept as far as the free margin of the gums, but if the gingival trough be examined by passing a delicate probe into it, a finely granular layer of subgingival calculus can be detected adhering to the necks of the teeth. Sometimes it takes the form of sharp nodules. Sometimes this local irritant is readily detected in larger quantities on the approximating surfaces of the roots of the teeth. Subgingival calculus is the active exciting cause of the trouble in these cases, and the process, although slower than the acute form already described, is just as sure in its destruction of the periodontal membrane and alveolus.

In these cases there is no loosening of the teeth, and sometimes the alveolar bone is enlarged over the roots of the teeth from the pathological action of a continuous slight inflammation. There is also a slight fetor from the region of the molars.

In the more acute forms, where marked inflammation is present, the tissues break down rapidly under the influence of bacteria, deep pockets are formed, in which pus collects. This may be confined to certain localities, where the inflammation has existed the longest, or it may be general. As the disease advances in severity and the pockets deepen by the loss of periodontal membrane and bone, bacteria become

largely responsible for the maintenance of the inflammatory process, which now extends deep into alveolar bone: the sockets become enlarged by the absorption of bone salts and a rarefying osteitis ensues, affecting the lamellæ, lacunæ and canaliculi of this transitory bony structure. The teeth become loose in their sockets, and every motion of mastication tends to increase the process of absorption set up by this new irritant which acts as a stimulant to the osteoclasts located in the proximity of the alveolar wall surrounding the roots of teeth. Pathogenic microorganisms enter the inflamed periodontal membrane and pass into the alveolar bone, where they exercise a deep-seated inflammatory action and are readily absorbed in large numbers into the general circulation. The system is able to resist this invasion of bacteria only to a certain point; beyond this immunity becomes lost and a general toxæmia often results. The toxæmic effect of microorganisms varies largely with the individual, and those previously or at the time affected by constitutional disorders, such as gastro-intestinal toxæmia, diabetes, neurasthenia, rheumatism, intestinal nephritis, anæmia or any form of toxæmia occurring in other parts of the system, would naturally lose immunity to the action of bacteria from the pyorrhœa source of infection much quicker than those who are otherwise sound. The effect of general toxæmia is undoubtedly that it aggravates the local affection and makes it more intractable. But constitutional disorders cannot be accounted the direct cause of the local infection.

As the disease advances to the last stage, when the teeth become so loose that it is a question whether they can be retained by any means (Fig. 3), when constant and copious discharge of pus indicates the severity of the affection, it will often be observed that the roots are smooth and entirely free

from calculus. This is not due to there never having been a deposit previously on them, but to the fact that the exudate of the first inflammatory process (from which subgingival calculus was first deposited on the upper portion of the roots) has been supplemented by infiltration of leucocytes and broken-down tissues mixed with microorganisms which constitute the pus, and this flowing constantly over the surface of the root gradually liquefies and dissolves those inorganic salts and carries them away, leaving the cementum smooth and clean.



FIG. 3.—Advanced pyorrhœa.

There are many other local irritant causes, besides those so far mentioned, which in some cases are the sole and only cause of the starting and maintaining of the disease, together with the bacteria, which are always a factor and fulfil the final destruction of the tissues concerned. They are often of a subtle nature and are readily overlooked in the search for the cause of the disease. They sometimes reveal no symptoms approaching the common forms of pyorrhœa, and their effect is principally on the periodontal membrane and the osseous structure of the parts. Prominent among these subtle causes is faulty contact points of the cusps of two or more teeth; this is brought about by the loss of one or more

teeth, and among the most obscure of these are cases of extractions in early life, which have apparently rectified irregularities of the teeth in a simple and sometimes apparently effective manner. An old practice of extracting the four first molars or the second premolars to overcome irregularities is sometimes responsible for this effect. Sometimes the effect can be traced to the loss of a single tooth, usually a first molar. This throws undue strain on certain teeth, which eventually results in a chronic irritation and inflammation of the periodontal membrane, accompanied with bacterial infection and resulting in periodontal disease, starting in the teeth affected.

Adenoids is another obscure cause by which mouth breathing induces the premonitory symptoms, at a very early stage, by impairing circulation in the gingival border.

Many other local irritant causes are to be found which produce more localized effects and provide a starting-point at the particular teeth concerned, the other parts of the mouth remaining normal. These include ill-fitting crowns or bridges, fillings with overhanging cervical margins or faulty contact points, abnormal spacing of the teeth, irregularities of the teeth, ill-fitting dentures, insufficient use of the teeth and the use of soft, pappy foods, these last two having a general influence on the whole denture. With all these local causes microörganisms are intimately concerned, and after a certain stage of advancement of the disease they may continue to maintain the progress of the disease even after the local irritant cause has been removed, giving the impression that pathogenic organisms are a direct cause of the disease.

CHAPTER II.

BACTERIA AND MOUTH PROTOZOA.

BACTERIA.

One of the most important factors which has to be taken into consideration in every case of periodontal disease is the bacteria which are associated with the suppurative inflammation characteristic of the disease. Microorganisms, which possess pathogenic properties, are always present in the oral cavity of healthy as well as impaired subjects. Whether pathogenic bacteria are capable of starting the irritation, which would lead to the establishing of the disease without the coöperation of any mechanical local irritant, is a question which does not seem to have been decided by authorities on the subject. Miller,¹ referring to this, says: "I am not able to form any decision regarding this matter, but so much is unquestionably certain, that the symptoms are greatly aggravated by local irritants, and that a removal of all irritations and extreme cleanliness are imperatively necessary in contending against this disease." It may be taken as an established fact, however, that whenever a slight irritant exists and has caused the appearance of the disease in a localized area of the mouth the pathogenic bacteria are capable of spreading the inflammation to adjacent parts and establishing the disease practically all over the mouth.

This fact is of great significance, because it points to the

¹ Microorganisms of the Human Mouth, p. 332.

important change which takes place in these microorganisms, which in healthy mouths are perfectly harmless, but in the changed environment resulting from existing disease are capable of becoming pathogenic organisms, exercising influence on neighboring healthy tissues and conveying disease to them. The establishing of disease from this source would be infinitely more marked in every case if it were not for the power of resistance with which healthy tissue is endowed, by which antibodies are produced to combat the bacteria; the toxic effect of the bacteria is overcome to a point of immunity, to which the tissues are capable of resisting, beyond this point it exercises an irritant effect on the tissues, resulting in inflammation, and organisms are able to multiply and dominate the tissue area affected by their presence in large numbers.

The degree of immunity varies considerably according to the power of resistance endowed in the tissues; thus it may be observed that some individuals retain a considerable amount of local irritant in very uncleanly mouths without developing pyorrhœa, while others develop the disease readily from the slightest amount of foreign matter about the teeth. In this respect environment, mode of living, occupation, heredity and constitutional disorders undoubtedly exercise great influence.

In the presence of the smallest amount of irritant in the gingival trough, which produces even a slight inflammation of the gingivus, it is possible that pathogenic bacteria are capable of entering the tissues and increasing in numbers, and although combated by the tissue-resistance to a point, local infection is inevitable, except there is an unusual immunity. It is therefore impossible to designate the stage at which pathogenic infection first takes place. It is erro-

neous to diagnose the disease as only starting when the gingival trough is deepened or pockets have been formed or pus is present. The early infection doubtless starts with the first slight inflammation and microorganisms begin then to adapt themselves to the tissues and to multiply in their effort to overcome immunity of the local area and to inflict their toxins on the tissues; from this stage onward, until immunity is lost and the tissues have broken down into deep pockets with pus, they are always present in ever-increasing numbers.

If this view of the early influence of microorganisms on the gingival margin were more universally accepted and acted upon the development of the disease in its advanced forms would be much rarer than it is at the present time, at least in individuals who visit their dentists at regular intervals.

A large number of different bacteria have been cultivated from pyorrhœa pus. Miller isolated twenty different bacteria from twelve cases of pyorrhœa, among which he mentions *Staphylococcus pyogenes aureus*, *Staphylococcus pyogenes albus* and *Streptococcus pyogenes*, "but was not able to determine the constant occurrence of any particular one which might be defined as the specific microorganism of pyorrhœa alveolaris." Miller's investigations appear to have been made from specimens taken from advanced cases, and in most instances he found but one kind of organism or one kind so predominated that the others could be left out of account. Later investigators describe the presence of many different kinds of bacteria very constantly present in the pus. Dr. C. P. Brown¹ found the almost constant presence

¹ New York Med. Jour., December 20, 1913.

of six kinds of bacteria in pyorrhœa pus, namely, *Bacillus influenzae*, the streptococcus, staphylococcus, pneumococcus, *Micrococcus catarrhalis* and diphtheroid bacilli. Goadby¹ investigated a large number of cases of the early stages of the disease and noted some difference in the microorganisms present at this stage to those found at more advanced stages; cultures made from them showed that the most commonly occurring were those corresponding to organisms of inflammatory catarrh: namely, *Micrococcus catarrhalis*, *Bacillus coryza segmentosa* or *Bacillus septus*, *Micrococcus pneumoniae* and in addition he found *Bacillus necrodentalis* (Goadby) and *Micrococcus citreus granulatus* (Freund). In later stages of the disease, when immunity of the general system was lowered and constitutional disturbances evident from absorption of toxins, the predominating organisms were usually staphylococci and streptococci or the pneumococci.

Microscopic examination of films of pus which are properly stained reveal a large variety of microorganisms (many of which do not grow on culture media), pus cells, hyaline cells, and epithelial debris. The bacteria include the numerous groups of cocci, bacilli, spirochetes and streptothrix.

MOUTH PROTOZOA.

Entamoeba buccalis has been found in the mouth from time to time and described by various authors. Gross, in 1849, described *Entamoeba gingivalis*, Sternberg, in 1862, *Entamoeba buccalis*, Grasse, in 1879, *Amoeba dentalis*, Flexner, in 1892, *Amoeba Prowazek* in 1905 *Entamoeba buccalis*; but it was not until 1914 that the researches of Chiavaro of Rome, and Barrett, of Philadelphia (simultaneously and

¹ Proc. Royal Soc. Med. (Odontological Section) iii, 55.

independently conducted), established the fact that *Entamoeba buccalis* is always present in pyorrhœa pus. The organism is described as measuring 6 to 30 μ , with a single vesicular nucleus of 1.5 to 4.5 μ in diameter, poor in chromatin, with a thick membrane. It is very mobile, constantly changing its form by thrusting out one, two or more lobular pseudopods in its activity and showing a clear distinction between the endosarc and extosarc. Chiavaro

1/2x



FIG. 4.—Large oblong and medium round type entamoeba with black masses ingested and surrounded by nourishing vacuoles. Fixed and stained with ferric hæmatoxylin of Heidenhain. (After Chiavaro.)

draws attention to the ingestion of bacteria by entamœbæ which could be seen in the protoplasm in fixed and stained specimens (Fig. 4).

Chiavaro came to the conclusion that “the entamœba has not a pathogenic action; on the contrary, as it feeds on bacteria, it is most probably an aid to the autodisinfection of the mouth.” This view the writer is inclined to accept after investigating 50 cases of pyorrhœa in which amœbæ

were found in 78 per cent., and in a paper read at the Royal Society of Medicine, June, 1915, expressed doubts, from a clinical standpoint, of the pathogenic action of *Entamoeba buccalis* in pyorrhœa. Barrett and Bass and Johns, however, pressed the claim, on clinical grounds, that *Entamoeba buccalis* being present "in a large proportion of pyorrhœa pockets and the disappearance of suppuration under appropriate treatment by a known amœbicide, such as emetin, have served to justify the claim that they are the immediate important factor in an overwhelming number of pyorrhœa cases."¹ This theory has been amply disproved and the presence of *Entamoeba buccalis* in the mouth is looked upon as having no pathogenic influence on the disease.

¹ Barrett, M. T.. Dental Cosmos, December, 1914.

CHAPTER III.

PATHOLOGY OF PERIODONTAL DISEASE.

THE pathological lesion exhibited in periodontal disease should be studied from its very initial stage when the physiological structure and function of the component cells are injured by some irritant cause and stimulated to excessive bioplastic activity which is always hyperplastic. The component parts of the structure under consideration must be recalled—the alveolar bone, the periodontal membrane and overlying fibrous or mucous tissue—all richly supplied with bloodvessels and containing terminal cells of singularly degenerative and transitory nature.

The initial pathological lesion is always instituted by an irritant in the gingival trough or injury to gingival tissue, resulting in inflammation, *i. e.*, dilation of bloodvessels, acceleration of blood flow followed by retardation, exudation and migration of leucocytes, stasis and degenerative changes, with accompanying proliferative and reparative changes. At the very earliest stage evidence of irritation to the gingival trough is hardly perceptible and can only be determined by clinical examination of the condition and contents of this space; but gradually this initial inflammation leads to marked congestion of the gingival fold and swelling of interdental papillæ accompanied by inflammatory exudates. A stagnation area is established in which mouth organisms are present; they begin to adapt themselves to favorable

conditions and produce an irritant influence on the tissues. The process of deposition of subgingival calculus now begins;

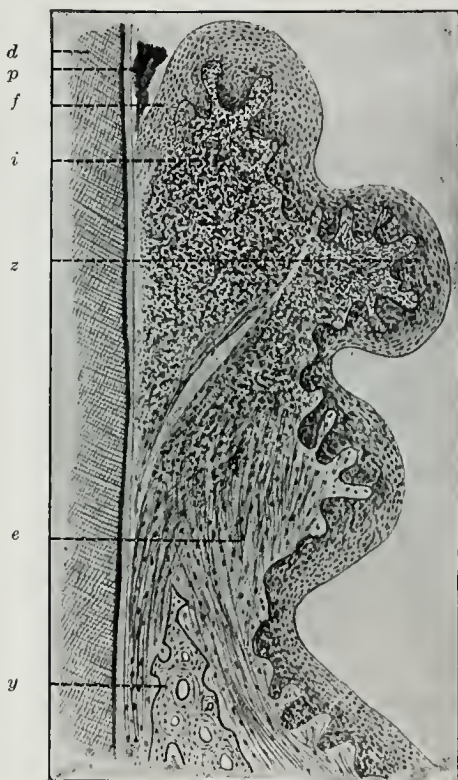


FIG. 5.—*d*, dentine; *p*, calculus; *f*, papillary layer of the gum; *z*, epithelial layer; *i*, tissue infiltrated with leucocytes; *e*, normal gum; *y*, normal bone. Magnified 80 times. Description from Dr. Znamensky's paper.

the terminal cells of the periodontal membrane subjected to degenerative and proliferative changes under increased nutritional conditions deposit the calcareous salts from the

blood by a process of pathological calcification much the same as that which governs other pathological calcifications,



FIG. 6.—A more advanced stage than that shown in Fig. 5. The destruction of the epithelium has commenced at *h* and *y*. Magnified 80 times.

“whether of the eye, the brain, the heart, the kidneys, the liver or the interna of arteries.”¹ This deposit takes the place

¹ Howe: Tr. Sixth International Dental Congress.

of the insertion of the periodontal membrane into the neck of the tooth and is constantly added to as cells become destroyed and exudation becomes prolific from increased hyperemia of the tissues. The destruction of periodontal attachment and epithelial lining of the gingival fold gradually deepens and widens the gingival trough, into which further irritants are readily introduced in the shape of food débris, providing a suitable pabulum for the maintenance and increase of pathogenic microorganisms.

The spread of inflammation to the alveolar bone sets up an osseous atrophy beginning at the free margin of the delicate alveolar structure into which is inserted the superficial fibers of the periodontal membrane; as the disease advances the destruction of periodontal tissue and consequent deepening of the gingival trough a space or "pocket" is formed. Osteoporosis often precedes comparatively slight inflammation of the alveolar border to a very disproportionate degree, hence the belief by some writers that osseous atrophy is the primary lesion in the disease; but in the author's opinion the osteoporosis and rarefying osteitis is an effect, not the cause, of periodontal disease.

Up to this stage no visible pus is present, the process of destruction of the tissues at the local initial site is admirably depicted in the accompanying illustrations by Dr. Znamensky¹ in which the calcareous deposit is seen in proximity to affected soft tissues.

Gradually the process of destruction of periodontal tissue and adjacent alveolar bone extends with a consequent deepening of the pocket into which accumulates destroyed epithelial cells, dead leucocytes, inflammatory exudations, food

¹ XVIIth International Congress of Medicine, London, Section XVII, p. 18.

débris and the resultant formation of pus which constitutes pyorrhœa alveolaris. The pathological process which has

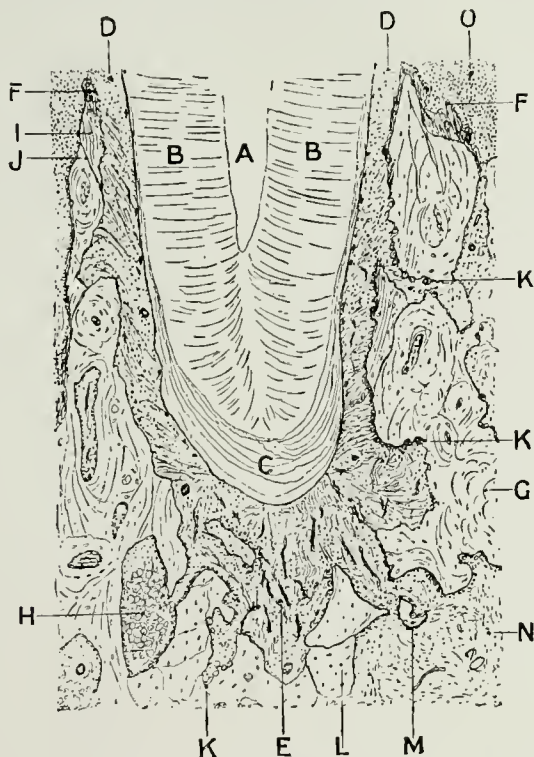


FIG. 7.—A, pulp cavity; B, dentine of tooth; C, hyperplastic cementum around apex of root; D, periodontal membrane, greatly thickened—hyperplastic; E, indifferent tissue at apical region greatly increased in amount; F, free edge of bone of socket becoming converted into fibrous intervening tissue; G, bone of socket presenting earliest signs of osteoporosis; H, large osteoporotic space in bone of jaw filled with bone-marrow; I, bone of socket partially decalcified and converted into osteoid tissue; J, junction of living with decalcified bone; K, osteoclasts producing lacunar absorption; L, bone of jaw only slightly altered by disease; M, sequestrum undergoing peripheral absorption; N, soft, cancellous tissue slightly changed from normal; O, inflammation of gum at neck of tooth. (From a drawing by Mr. A. Hopewell-Smith, *Lancet*.)

preceded the formation of pus differs in no way from the severe consequence resultant from its uninterrupted continuance, except in the severity which now exhibits itself in a marked osseous lesion. The weakened tissues become a prey to infection of pathogenic microorganisms, which overcome the defensive powers provided in the tissues by which leucocytes and tissue cells produce antibodies to combat their invasion. Great changes take place in the alveolar bone as the inflammation extends along the Haversian canals. Hopewell-Smith¹ describes it thus: "the bone becomes transformed into an osteoid tissue through loss of its calcium salts, then passes into an intervening fibrous tissue, and finally is attacked by the inflammatory exudation and cells, its bays and recesses become meanwhile greatly enlarged and filled with loose soft tissue, and the Haversian canals enlarged and irregular, the condition being termed osteoporosis."

These pathological changes which finally bring about the loosening and exfoliation of the teeth, are purely local (with a local irritant cause to start with), finally involving the entire transitory bony frame supporting the teeth; the process of inflammation becomes chronic but never ceases unless the local irritant cause is completely removed. The rapidity of advance of the disease, its fluctuating severity, the variety of forms it apparently assumes and its intractability to treatment appear to be dependent chiefly on the structure of the alveolar bone. The degree of development and density of this cancellous osseous tissue has a distinct influence on the resistance it is capable of exerting against inflammatory action started in direct proximity to its most vulnerable

¹ Dental Cosmos, 1911, p. 405.

part (the margin). Examination of museum specimens clearly indicates that pathological changes take place more extensively in specimens which exhibit lack of development of alveolus, and that resistance to the disease is evident in well developed jaws.

The present-day mode of living is liable to adversely affect the development of the bone around the teeth, which are not called upon to perform their normal functions, and as a consequence a fragile alveolar structure is formed about the roots of the teeth which is incapable of resisting attack by disease. An almost distinct form of pyorrhœa exists, which, if studied closely can be traced to this lack of development; the jaws are usually thin (often in keeping with a delicate osseous frame), the outer plate of alveolus is exceedingly fragile, consisting of a thin, pointed film of bone at the margin about the necks of the teeth; the muscles of mastication are correspondingly ill-developed, suggesting lack of proper masticating exercise. The slightest irritation to the muco-periodontal membrane affects this delicate osseous structure and starts osteoporosis which advances in great disproportion to the amount of inflammation present, causing a considerable loss of bony structure before any sign of pus is present; the overlying gum tissue is universally crimson to a point corresponding to the advancing inflammation in the osseous structure. Pockets of considerable depth rapidly form about such teeth, and they loosen very rapidly. This form of pyorrhœa is often attributed to constitutional disorders, but is only indirectly attributable to this, inasmuch as a weak osseous frame about the teeth is readily affected by inflammation.

On the other hand if the alveolar process is well developed and the maxillary bones normal in size, osteoporosis and

rarefying osteitis of the type described is never present. Pyorrhœa may develop as a consequence of irritants accumulating in the gingival trough, but the progress of the disease is slow and marked rarefying osteitis does not occur, until the last stage of the disease is reached.

The disease, when accompanied with conditions of general disease of the osseous system, is undoubtedly influenced by pathological conditions peculiar to those osseous diseases, but those cases are comparatively rare, and have special significance only on the pathology of those particular cases.

The pathological reason given for the theory that periodontal disease is purely local is amply verified by comparative studies of the disease in animals. Colyer¹ states that in the horse "the initial lesion was shown to be a slight destruction of the interdental papillæ, probably from the injurious character of the food." In cats and dogs he says "the explanation is that soft diet clings about the teeth, stagnation areas are formed, and a marginal gingivitis is produced." It is certainly necessary that a local irritant cause must be present to disturb the normal physiological function of the tissues and produce the initial pathological lesion. Talbot² has shown by experiment on dogs that pyorrhœa could not be produced by injecting pyorrhœa pus into healthy gum tissue, but infection occurred when tissues were inflamed. "The pathologic finding in these cases were not unlike inflammation and infection in other tissues."

The most frequent initial irritant in periodontal disease in man is undoubtedly stagnation produced in the gingival trough as a result of modern diet. Particles of soft sticky food adhering to the necks of teeth produce the initial irrita-

¹ Chronic General Periodontitis, p. 46.

² Interstitial Gingivitis, p. 126.

tion, and start the inflammatory stagnation area which leads to more extensive inflammation, and ultimately to breaking down of the component structures of the parts involved.

This conception of the pathology of periodontal disease condemns the term "pyorrhœa alveolaris" because the disease exists in all its phases before pus is present, and the pus stage is only the result of continued pathological process, which should be recognized in the earlier stages.

The influence of constitutional infectious diseases, such as syphilis, tuberculosis, actinomycosis, etc., or constitutional irritants, such as drugs, metal poisoning, auto-intoxication, is undoubtedly reflected in the intractability of periodontal disease, when that disease has been started by a local pathological lesion, but these cannot be said to cause the disease except when they are capable of producing interstitial gingivitis, as in the instance of drugs or metal poisoning, otherwise, a perfectly healthy gingivus may be maintained in the presence of constitutional disorders.

CHAPTER IV.

TOXÆMIC EFFECTS OF PERIODONTAL DISEASE.

ALIMENTARY toxæmia due to pyorrhœa pus is one of the commonest effects of the disease, and, as the term implies, covers a very wide field. There are many toxæmic effects on various parts of the body which can be traced directly to the local infection. Microörganisms from this source enter the system by direct absorption into the blood stream at the source of infection and by swallowing of pus.

The structure of the alveolus and the attachment of the teeth by periodontal membrane exposes these parts in a singular degree to violence, with the minimum of protection. Inasmuch as this is the only instance in the body where periosteum is exposed at the surface with little or no protection, there being only the gingival fold and a thin epithelial layer overlying, it is therefore prone to injury and infection, and when infected, being in immediate contact with bone tissue, becomes a septic ulcer more violent and more toxic than ulcers in gastro-intestinal tracts, which have some detoxicating organ, like the liver, to correct and modify the toxins introduced from this source into the blood. Absorption from this source is therefore direct, profuse and far-reaching.

The supply of streptococci and other organisms from pyorrhœa when pus is swallowed passes over the tonsils and pharynx into the gastro-intestinal canal, where in passing

it often produces different stages of tonsillitis, pharyngitis, gastritis, intestinal inflammations, gastric erosions and ulcers in various parts of the alimentary tract, and appendicitis. Of 150 cases recorded by Goadby,¹ 42 per cent. had gastro-intestinal affections, 56 per cent. had rheumatic symptoms, peri-articular arthritis, arthritis deformans or fibrositis and muscular rheumatism.

Toxins absorbed from oral sepsis are the sole cause of many inflammatory diseases of the eye, although the nature of the poison does not seem to have been discovered. Wm. Lang² has pointed out that the removal of the pyorrhœa source of sepsis has been sufficient to reduce inflammation and pain in the eyes, and asserts that "in recent cases of central choroiditis the lost vision quickly returns," after the pyorrhœa has been cured. Mr. J. B. Lawford,³ in summing up the influence of oral sepsis on the eyes, says: "The recurrence of iridocyclitis with fresh outbreaks of pyorrhœa leave little room for doubt that the ocular lesions are caused by the septic condition of the mouth, and probably through the agency of toxins."

Neurasthenia is a common result of toxic poisoning from oral source. The characteristic symptoms of malaise and fatigue precede the more marked fatigue neurosis, which is often accompanied by thrills and wavy feelings and also insomnia. Toxins carried into the blood stream account, in a measure, for neurasthenia; but there is also the inflammatory irritant at the local site of infection, having a direct influence on the vasoneural circuit by which short-circuiting of nerve energy is brought about and the perpetual abnormal closure of the sensory circuits produces constant ganglionic

¹ Proceedings of Royal Society of Medicine, vi, Part I, p. 224.

² Ibid., p. 301.

³ Ibid., p. 124.

discharges, resulting in fatigue of nerve energy, which is only relieved by rest and the removal of the irritation. The removal of this septic and irritating influence by the cure of periodontal disease in these cases has so frequently brought about complete change in the neurasthenic condition that the writer has no hesitancy in expressing his conviction that periodontal disease is often the sole cause of the nervous affection.

The degree of infection which is likely to bring about toxæmic effects on the system deserves close attention. It is generally conceded that a large amount of sepsis in the oral cavity, extending over a long period, is undoubtedly a frequent cause of constitutional disorders; but observation of many cases which have been noted has convinced the author that very slight gingival infection, of comparatively short duration, is capable of producing the same effect. The reaction of the tissues by which a protective barrier is set up against the toxins produced by sepsis depends on the inherent vitality of the tissues, that is, on their functional activity in producing antibodies at the local site to combat the microorganisms—but this protection is often insufficient in cases of only slight infection to provide immunity against toxæmia, which may result when only a slight gingival affection apparently is present without the presence of pus.

Absorption of toxins from apparently slight pathological lesion of the periodontal membrane producing constitutional disorders identical with well known effects of extensive pyorrhœa alveolaris may be illustrated by instances in actual practice recorded in the following typical cases:

CASE I.—Mrs. G., of Oxford, aged forty years. The condition of the gingival margin only revealed a slight gingivitis; the teeth were perfectly clean above the gingival margin, but in

the gingival trough, clinging to the necks of the teeth, a rough granular deposit of subgingival calculus was detected and the bleeding from the gingival fold was profuse. There were no pockets, only a deepening of the trough, which also contained some food débris, no pus on pressure. Constitutional disorders complained of were malaise, rheumatic pains in the knee- and shoulder-joints and headaches. The patient was in the habit of taking her own temperature, which she said was usually 100° to 101° at night, except for a few days after visiting her dentist and "having a good clean up." Her dentist, who referred her to me, rightly said there was no "pyorrhœa;" her medical adviser could find no cause for temperature and other symptoms. Treatment consisted in thoroughly removing all calcareous deposits and electro-sterilization of the gingival trough with zinc ions. Temperature disappeared after the third treatment, malaise and headaches in a fortnight and every trace of rheumatism in a month. Perfect health has followed ever since.

CASE II.—Mrs. B., aged forty-two years, seemed to have perfect condition of gums and teeth, no redness or bleeding until an instrument was passed into the gingival trough; no pus or deep pockets. The superior first premolars had been extracted "to make room" (Fig. 8). This had upset the normal balance of the articulation producing undue stress on the superior lateral incisors and second molars resulting in chronic irritation of the periodontal membrane, some rarefying osteitis about the roots of these teeth, and slight loosening of the teeth. A crust of subgingival calculus existed on all the teeth and in larger quantities about the loosened teeth. Radiograph (Fig. 9) shows the condition of the alveolus about the incisors. The patient complained of malaise and rheumatic pains in the joints. Removing

the calculus and relieving undue stress, with a course of electro-sterilization with zinc and iodine ions, particularly



FIG. 8.—Showing abnormal occlusion due to extraction of superior first premolars.

about the loosened teeth, produced a complete cure of rheumatism in six weeks, and there has been no recurrence of constitutional symptoms.

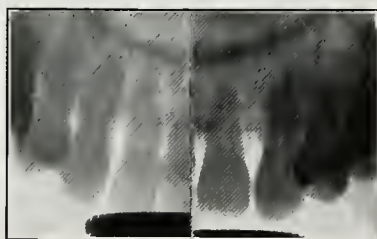


FIG. 9.—Radiograph of superior incisors.

Very severe rheumatoid arthritis may be brought on by apparently slight periodontal disease in what may be

described as well kept mouths, as illustrated by the following case, which was reported by the writer at the Royal Society of Medicine, Odontological Section, July, 1918.

CASE III.—Mr. B., aged about forty-five years, consulted me on June 28, 1917. He was unable to walk and could only move about with assistance and the aid of crutches; his knee-joints were very much bent and perfectly rigid. He had been in that condition for a considerable time and informed me that the doctors in attendance attributed his arthritis to septic absorption, the only source of which was his mouth, and that there was a question as to the advisability of extracting all his teeth, which were perfectly sound and not loose. He had been treated for pyorrhœa by vaccine therapy, the predominating organism being the streptococcus from which the vaccine was prepared; but this appeared to have had very little effect on his arthritis, although the state of his gums seemed to have been much improved.

His teeth were perfectly clean as far as the gingival border, the gums looked fairly healthy, there being only a fringe of redness about the lower incisors and a purple hue about the lingual surfaces of the molars. The patient worried over his condition a good deal; he brushed his teeth several times a day and used peroxide of hydrogen freely. There was no visible pus anywhere. Examination of the gingival trough revealed the true state of things. There was a hard brown crust of subgingival calculus encircling the roots of the teeth from which the periodontal membrane had receded, forming shallow pockets. These pockets were singularly free from food débris. The irritant here was the calculus, which had kept the gingival fold and periodontal membrane in a state of inflammation, as was evident from the bleeding, which occurred when an instrument was passed into the trough.

Tissues in this condition become readily infected, the toxins passing into the system by direct absorption into the circulation.

Treatment consisted in removing every particle of calculus and polishing the root surfaces. At each sitting ionization of the gingival trough and alveolus was carried out with zinc ions, the patient being a good electrical subject, a current of 5 to 10 ma. was tolerated and the tissues yielded immediately to the treatment.

On August 10 he was discharged, every sign of inflammation in the gingival trough having disappeared. Three months later the pockets were examined and found to be in the same healthy state. A decided improvement had taken place in the rheumatoid affection; the patient could walk across the room without crutches; although his knees were still bent and stiff, he nevertheless expressed his conviction that he was progressing favorably. On June 10, 1918, when he was last seen (ten months after treatment), the gingival trough was perfectly healthy; he had long since discarded the use of crutches, his knees were almost normal in shape, only a slight stiffness remaining; he was able to walk long distances and had resumed his occupation as an engineer. He had had no other treatment since his course of ionization; had simply kept his teeth clean, using a benzoic acid and thymol wash on the brush twice daily.

There can be no doubt that the rheumatoid arthritis was caused by this apparently slight septic inflammation of the gingival trough, and a cure was effected by removing the local irritant cause, and at the same time sterilizing the affected tissues by zinc ionization—both being necessary in my opinion.

Passing to the effects produced by oral sepsis in advanced

cases of periodontal disease, when in addition to direct absorption of toxins at the site of local infection large quantities of pus pass into the stomach of what may be an already weakened constitution, the presence of a great and constant supply of organisms tends to destroy the gastric defence and weaken the tissues, making it possible for organisms to pass into the intestinal tract. Under general weakened conditions of the intestines they are able to enter the system, poisoning the tissue cells by their toxins, and bringing about structural changes and disease.

The germs associated with oral sepsis vary and systemic conditions vary, so that the absorption of toxins produces a wide variety of diseases—in some arthritis, in others anemia, in others neuritis and so on, according to favorable conditions for inhibition of the microbes.

The pathological significance of oral sepsis has often led medical diagnosis astray and caution is necessary in deciding that other diseases are caused by mouth bacteria, which may not be the direct source of infection; nevertheless, the oral condition should always be eliminated without too definite promise of eradication of the disease.

Sufficient importance does not appear to have been placed on the fact that spirochætes are largely associated with pyorrhœa; apart from the knowledge that they are usually present, the subject does not seem to have engaged the serious consideration of dental pathologists in relation to periodontal and general disease. Moritz has described the presence of spirochætes in the tissues and bone-marrow in a case of anemia. J. G. Thomson and Dr. Thomson¹ have made extensive investigations of this subject and they state

¹ Beck, Marcus: Laboratory Reports, i, 65.

that "If medical men in this country would begin a systematic examination of the mouths of patients, we have not the slightest doubt that, like ourselves, they would be astonished to find millions of spirochaetes in small scrapings from the alveolar margin. These in many cases are too numerous, even if they are considered only saprophytes, to be associated with good health. They must be elaborating toxins of a peculiar character and they must also assist in the general destruction of the tissues in the alveolar margin."

It is significant that in trench mouth, which was brought into prominence during the great European War, spirochaetes are found to predominate so greatly that they are generally accounted responsible for this pathological condition; further, the systemic changes which accompany the disease clearly indicate that the organism finds its way into the tissues and alimentary tract and finally becomes so altered as to be able to live in the blood stream. The enormous increase in numbers in cases of trench mouth, pyorrhœa alveolaris, alveolar abscess and Vincent's angina must produce toxins detrimental to health and in a great measure be responsible for the constitutional disturbances usually present in these diseases.

CHAPTER V.

EARLY DIAGNOSIS OF PERIODONTAL DISEASES.

Too much stress cannot be placed on the importance of early diagnosis of periodontal disease, the incipient stage of which is far too often overlooked so that the disease, which has its origin in the gingival trough, is allowed to progress beyond the early infection of periodontal tissues, before it is recognized as of any importance. The gingival margins, when normal, are of tough bloodless tissue, of a clear pink hue, capable of resisting considerable pressure from tough foods during mastication without discomfort; redness, tenderness, or bleeding are clearly indicative of beginning inflammation, the causes of which should be diagnosed before it reaches the stage of acute congestion. Should any signs of abnormality be present, a minute examination of the gingival trough should be instituted, when invariably some foreign substance will be revealed, which accounts for this stagnation area in the tissues leading up to what will result in gradual deepening of the trough and breaking down of the thin epithelial layer of tissue overlying the poorly protected bony structure immediately underneath. The utmost importance should be placed on these signs, as the beginning of disease, and proper treatment advocated to avert the consequences of periodontal diseases, which almost invariably follow.

Passing from this early stage, symptoms are more easily

diagnosed, but even then they are far too often looked upon as a gingivitis with no direct bearing on ultimate periodontal disease. The heaping up of gum tissue, congestion with free bleeding on brushing or touching the gums (there being no visible pus) should be included in the possibility of septic infection having started in an unhealthy gingivus, in which a deepened gingival trough and damaged periodontal attachments, under influence of local irritants, will continue to break down until pus supervenes.

In well kept dentures, where daily hygienic measures have been taken, often none of the hyperemic symptoms are present, still a wasting of the gum tissue is evident with absorption of the alveolar bone, elongated necks of teeth are



FIG. 10.—Examining scalers.

present with disappearance of interdental papillæ. In these cases examination of the roots of teeth made by passing a delicate hooked scale (Fig. 10) parallel with the long axis of the tooth, and scraping the surfaces under the thin taut overlying gum, will reveal a granular deposit of calcareous salts, firmly adherent to the surfaces of the roots. Often this irritant is only to be found on the surfaces of approximating roots in the form of sharp granules (Fig. 11) readily discernible by a scaler but often in the form of a dark hard crust, smooth on the surface and most tenacious, requiring considerable force to detach even a small bit of it. This condition should be recognized as slowly progressing periodontal disease, in which pathogenic microorganisms play

an important part. It is not caused by overbrushing as often designated.

Acute and chronic periodontal disease (pyorrhœa alveolaris) which is unmistakable, in which deep pockets are established, filled with decomposing food, epithelial débris and pus, with great loss of bony tissue and loosening of teeth, is but a continuation of the early stages described, which have progressed unrecognized by patient (and often by dentist) for many years before reaching the climax, which brings it into prominence.

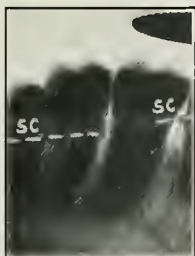


FIG. 11.—SC, subgingival calculus.

The calcareous deposit referred to so far is by no means the sole diagnostic feature, many other causes require diagnosing, some of which are of a subtle nature requiring infinite care to recognize. Among these, faulty occlusion and undue stress are of importance. The common practice of extracting premolars or first molars to relieve “overcrowding of the arch” in the young, almost invariably leads to disturbing the balance in certain areas of the denture, as well as the creation of abnormal spaces, which results in time in periodontal disease. The loss of teeth from caries, usually the molars in either maxilla or mandible, especially when this is unilateral, and their place is not supplied with artifi-

cial substitutes, is a fruitful means of producing undue stress on other parts of the denture and supplying an irritant to the osseous frame and fibrous attachments of the teeth, by which periodontal disease can be maintained.

The palatal and lingual surfaces of the gingivus are frequently the sites of infection, when artificial substitutes for teeth impinge on these parts; most frequently is this noticeable in the mandible when the molars are lost and a plate is

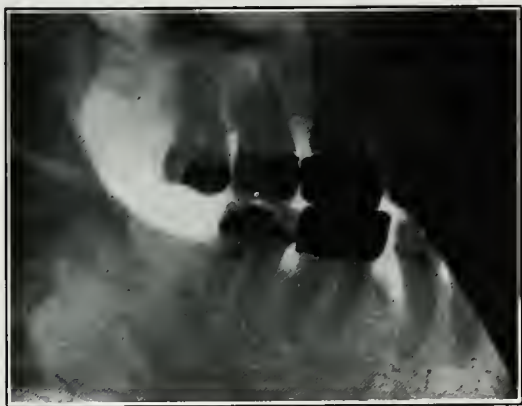


FIG. 12.—Badly constructed shell crowns.

made to rest on the lingual surfaces of the incisors, absorption of the alveolus at the back allows the plate to settle down snugly on the front portion and provides a constant irritant acting injuriously on the gingivus and underlying bone. No form of treatment will succeed while this exists.

The faultily constructed shell crowns or banded crowns, where a space exists between the free edge of the metal and the root is an undoubted source of infection, which requires careful investigation, not that every crown of this description

is necessarily a cause of infection, far from it, but it should be determined whether the free edge of metal is so well adapted to the circumference of root as to preclude any irritating influence on the gingival fold, either directly by pressure, or indirectly by retaining foreign matter in the space. Should congestion about the edge of a crown be present, it is necessary to determine whether faulty construction is responsible, or merely the spreading of infection from adjacent parts, accompanied by a general irritant cause, such as calculus and stagnant food. The same applies to bridges and all forms of fixed appliances. It is often necessary to condemn these as a source of periodontal disease; but when properly constructed, in the mouths of patients who are capable of carrying out proper daily hygienic methods, there is no reason why they should be condemned at sight, even when pyorrhœa symptoms are present in other parts of the mouth; and here it becomes necessary to determine the true cause of any inflammatory symptoms present and decide by accurate diagnosis the advisability of retaining a fixed appliance in the face of existing periodontal disease when treatment is to be undertaken.

In the undertaking of treatment of periodontal disease it is of utmost importance to recognize the irritating nature of overhanging fillings, and faulty contour of fillings (Fig. 13). This source of irritation, if not removed, will cause failure to preclude anything like permanent results by treatment, and recurrence, even after apparently good results, is unavoidable at those parts. Here radiography is invaluable in arriving at a sure diagnosis. A slight ledge may be detected by this means, which entirely escapes the exploring instrument. Inversely the existence of caries at or under the gum level provides an irritant cause, which requires detection,

not only does the sharp edge of a cavity in contact with the gingival tissue produce inflammation, but the contained débris of decay and decomposed food provide a perfect



FIG. 13.—Faulty filling.

pabulum, on which microörganisms thrive and keep up the source of infection.

Radiographs should be procured of all advanced cases of periodontal disease, without which it is difficult to diagnose the extent of the disease. It is important to determine the



FIG. 14.—Imperfect crown causing periodontal disease.

amount of destruction of bone, the depth of pockets, the condition of the bone, the presence of irritants such as subgingival calculus, faulty fillings or crowns (Fig. 14). On the

condition revealed a diagnosis can be made of the teeth which should be extracted. Many teeth which clinically appear hopelessly involved will present in the radiograph a healthy

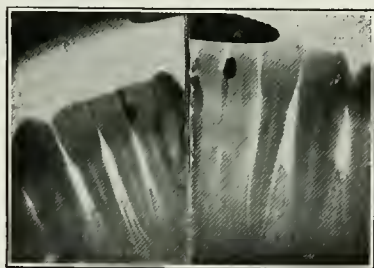


FIG. 15.—Enlarged sockets due to undue stress, with well defined linea dura.

alveolus, with a well-defined linea dura about sockets, which are enlarged in consequence of undue stress. Such teeth can frequently be saved.



FIG. 16.—Subgingival calculus on roots.

The presence of subgingival calculus is readily revealed when it exists on a surface of the tooth forming a shadow by the rays in the radiogram (Fig. 16), and when seen is usually of the hard dark variety, which is most difficult to remove.

Faulty fillings and defective crown work are readily detected, and the condition of the bone in the latter is a guide to determining the advisability of recrowning in the presence of existing periodontal disease.



FIG. 17.—Faulty filling.

Valuable diagnostic information is afforded by correct reading of the condition of the bone, as shown in radiographs, by which the degree of absorption and rarefying osteitis can be accurately judged. If a perfectly defined linea dura is shown it would exclude to a great extent the probability of

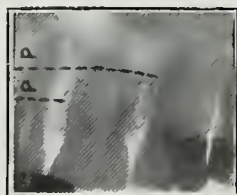


FIG. 18.—a, well defined linea dura.

osteoporosis and infiltration of microorganisms deep into the bony structure, but if every trace of the linea dura has disappeared the bony structure presents a homogeneous radiolucent appearance, extensive rarefying osteitis with general bacterial infection can be diagnosed, which justifies

the opinion of long standing periodontal disease of a serious character. So, too, an enlarged socket about the root of a tooth, where bony tissue has not absorbed to a great extent

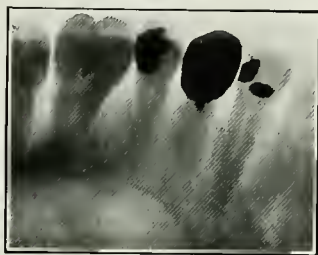


FIG. 19.—General rarefying osteitis.

points to the possibility of undue stress causing movement of the tooth in its socket, and often gives the clue to this obscure cause of irritation and loosening of the tooth (Fig. 20).

Diagnosis of the condition with relation to other existing pathological lesions, when it becomes necessary to determine the advisability of retaining teeth in spite of suspected toxic

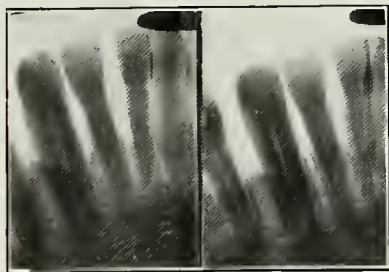


FIG. 20.—Linea dura affected by movement of teeth in their sockets.

influence of organisms on the system, requires precise judgment of the extent of the disease and as to the possibility of retaining certain teeth in a functional and healthy condition

after the most careful treatment. It must be borne in mind that conservative treatment is aided by the removing of the septic source, by improvement in the general health and with it the tissue at the local site of infection, so that the necessity for extracting every tooth from which pus exudes or may be expressed from its socket, does not apply as a hard and fast rule, but teeth, which after treatment cannot possibly be kept clean by the daily hygienic efforts of the patient, should at once be extracted, such as molars, when the alveolar bone has absorbed to such an extent that the bifurcation of roots is exposed, forming a receptacle for lodgment of food. Teeth which have pockets extending to the apices, when, on passing a probe, necrotic condition of the root can be felt, should be extracted.

Dead teeth, with chronic apical abscesses, are more to be feared in relation to constitutional disorders than the worst pyorrhœa conditions, and no tooth should be retained under those circumstances, unless the operator is convinced of a radical and speedy cure of the lesion by surgical methods.

In the molar region, advanced pyorrhœa, with deep pockets between the teeth, which, if the disease is checked by treatment, is liable to recurrence, it being beyond the patient to keep the space clean, and in view of toxic effects, the extraction of the middle tooth, that is the second molar, often checks the disease about the two adjoining teeth, conserving those without fear of a potential source of infection.

In general it should be determined at the very outset what sources of infection exist which cannot be totally eliminated by treatment and proper after-care, and these should be removed at once, whether they be in the nature of crowns or bridges or hopelessly involved teeth, for fresh attacks aggravate the pathological lesions in other parts of the body, which do not then respond so readily to treatment.

CHAPTER VI.

TREATMENT OF PERIODONTAL DISEASE.

THE treatment of the disease in this work is based on the etiology and pathology given, and definite lines are pursued systematizing the *modus operandi*, details of which will be gone into.

For convenience of description, the different stages of periodontal disease are here classified as follows:

1. Incipient infection of the gingival trough.
2. Septic infection of the gingival trough without suppuration.
3. Chronic septic infection of the periodontal membrane without visible suppuration—"dry pyorrhea."
4. Acute septic infection of the gums and periodontal membrane without visible pus.
5. Chronic periodontal disease with pus. "Pyorrhœa alveolaris."

1. Treatment of Incipient Infection of the Gingival Trough.
—Special reference has been made to this stage of periodontal disease in writing on etiology, pathology and diagnosis, with the intention of emphasizing the importance of preventing the development of pyorrhœa alveolaris by the adoption of simple methods of treatment. Prior to the stage requiring any treatment, a good deal can be accomplished by recognizing existing conditions, which are likely to promote periodontal disease in later life, and by advising parents what to do.

In this respect it is of course necessary to obtain early control of the child. Such conditions as poor development of the oral structures, due to modern changes in natural and primitive environment, and absence of normal functional activity of the muscles of mastication, due to dietetic arrangements of modern life.

Mothers should be advised that soft pappy foods of starchy character should be avoided, and a certain amount of fibrous hard food and fresh fruit introduced into the diet, following out the principles laid down by Dr. Sim Wallace,¹ Dr. Harry Campbell and others. Mouth-breathing and adenoids should be attended to. Irregularities of the teeth can often be easily rectified by early expansion of the arches, as pointed out by Dr. Northcroft. Intelligent training of the child in proper hygienic methods should be undertaken. All these will do much to prevent the development of periodontal disease.

When incipient infection of the gingival tissues has already been established, whether in children or adults, treatment consists in removing every particle of irritant on the tooth surfaces and in the gingival trough, and polishing these surfaces to the highest state of perfection. The gingival trough if congested, should then be treated by electro-sterilization. A platinum electrode should be wrapped with a few shreds of cotton-wool, saturated with a 10 per cent. aqueous solution of tincture of iodine, this should be passed into the gingival trough and the current gradually turned on from the negative pole, the patient holding the positive electrode; 1 or 2 m.a. current should be passed while the operator slowly moves the electrode in perfect contact around the necks of the affected teeth with a wiping motion

¹ Modern Dietetics in the Causation of Disease.

of the instrument, which will medicate the gingival trough, as well as remove any particles of débris which may remain after the polishing process. This treatment should be carried out wherever any redness or inflammation of the gingival border exists; a single treatment will often be sufficient to restore the gingivus to a normal condition, but in case inflammation persists in any isolated part, the process should be repeated there, after further cleansing and polishing precautions have been taken. The gingival trough once enlarged by the entrance of foreign substances is liable to recurrence of the trouble, and the patient should be warned that in future any bleeding of the gums during brushing is an indication of recurrence, when treatment will again be necessary. Advising the use of fresh fruit after the last meal of the day, especially biting into an apple, is one of the potent means of cleansing the necks of the teeth from sticky carbohydrate foods, which comprise the chief danger in incipient periodontal affection.

2. Septic Infection of the Gingival Trough without Suppuration.—This stage of periodontal disease follows sharply on the preceding, and is universally looked upon as simple gingivitis. Congestion of the gingivus is marked, with loosening of the gingival fold, and deepening of the gingival trough, from which bleeding readily occurs on pressure. A process of breaking down of the gingival fold, the alveolar bone and superficial fibers of periodontal tissue is established, and although no pus is present, pathogenic organisms inhabit the tissues, which are in a condition to facilitate their growth and maintenance.

Examination of the necks of the teeth will invariably disclose irritants in the form of calcareous deposits and stagnant food. Treatment consists in thorough instrumenta-

tion, removing every particle of foreign substance and polishing the surfaces of the teeth. Bleeding and tenderness of the gums will often interfere with accomplishing this end satisfactorily at the first operation, but ionization should be carried out then, and the case seen again in a few days, when invariably marked improvement will be noted, and the completion of instrumentation and polishing facilitated, Ionization with zinc ions should be carried out by passing a

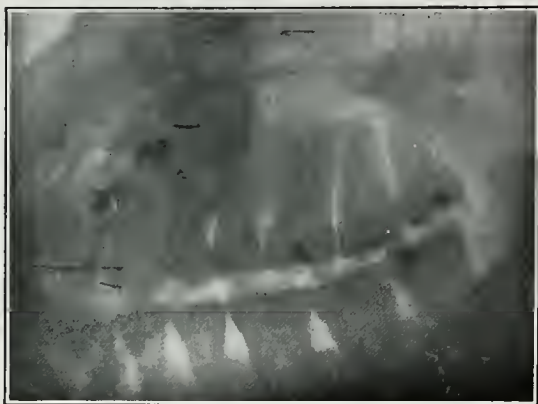


FIG. 21.—Progressing periodontal disease.

spear-shaped zinc electrode of large enough size to readily pass into the gingival trough and wound at the point with a few shreds of cotton-wool saturated with 3 per cent. zinc chloride, the electrode should be steadily held in position, the patient holding the indifferent electrode. The current from the positive pole should then be turned on gradually, with the object of passing 2 or 3 ma. at least, if this amount can be tolerated. The electrode should be moved very slowly, in perfect contact around the necks of the teeth with a wiping

motion, taking about half a minute to pass from interspace to interspace on the outer surface of each tooth, but here discretion must be exercised—parts obviously worse require more time at the expense of parts less affected. The saliva should be kept away with cotton swabs, and the zinc chloride replenished from time to time during the operation, turning off the current each time before removing the electrode.

The case should not be discharged until it has been ascertained that all inflammation has completely subsided, and the gums have resumed their normal appearance and toughness, reëstablishing nature's barrier to the introduction of food débris into the gingival trough. The patient should be carefully instructed in an effective method of daily hygiene of the mouth, it being certain that the cause of the trouble is attributable to ignorance or neglect.

3. Chronic Septic Infection of the Periodontal Membrane without Visible Suppuration—"Dry Pyorrhœa."—This phase of the disease, which has been described from etiological and pathological standpoints occurs in well kept mouths, and presents many difficulties in treatment, principally on account of the subtle, slowly progressive nature of the affection. The disappearing interdental papillæ and taut wasted gums resting on exposed necks of teeth, which may be perfectly polished as far as the gingival border, effectively conceals an etiological factor in the form of calcareous deposits closely adherent to the roots of the teeth beneath the gums. This irritant takes on various forms, from hard sharp nodules to a finely granular layer. The treatment, to be effective, imposes on the operator most delicate and exacting instrumentation. Thin, sharp, hook-shaped scalers, with rigid shafts, should be passed between the gum and tooth surface to the bottom of the gingival trough, parallel with the long axis of the tooth,

and with a hooking or rather planing motion, scrape off or plane off every particle of foreign matter, erring on the side of removing a thin layer of cementum rather than leaving any deposit adhering to the tooth, considerable force being often necessary to accomplish this. The author's set of scalers if well made with small planing blades are very effective in carrying out this planing of the roots. Further polishing of the root surface should be effected with thin wood points and pulverized pumice made into a paste with 20 per cent. aromatic sulphuric acid. In addition stiff cup-shaped brushes



FIG. 22.—Sturridge's scalers.

used with the pumice paste assist in removing tartar and polishing the surfaces about the gingival border.

Instrumentation and polishing having been thoroughly carried out, it remains to deal with the pathogenic micro-organisms, which constitute an important etiological factor. A thin platinum or zinc electrode should be wrapped with a few shreds of cotton-wool saturated with 3 per cent. zinc chloride and passed into the gingival trough, the current from the positive pole turned on gradually, the patient holding the negative electrode. If the teeth are very sensitive

0.5 m.a. current may be all that will be tolerated in the incisor region, but the molar region may admit of considerably more current—2 to 5 m.a. The electrode should be slowly moved parallel with the long-axis of the tooth around the neck, with the object of imparting a sufficient dose of ions to sterilize the tissues. In areas where the tissues are much wasted and very thin, the time required to effectively impregnate them with ions is very much less than in thicker areas like between the molars; one quarter of a minute to half a minute should be occupied in moving the electrode about the neck of a tooth, varying the time in direct ratio to the thickness of the tissue and current strength in use, that is, if the tissues are very thin and 3 m.a. is in use, time required will be one-third that if only 1 m.a. is tolerated. Similarly in thicker tissues the same current strength will require more time to secure proper depth of penetration of ions.

The number of treatments required depends upon the severity of the case and the current strength available. Three the first week, two the second and once a week after, until every sign of inflammation about the gingivus has disappeared, gives some idea of the course that should be pursued. Fetid odor is often the only symptom complained of by the patient, until this disappears entirely the treatment should be vigorously continued every second day with as strong a current as possible and extended time, in any particular region from which the patient locates this symptom; the location is usually between the molars.

Restoration of lost bone and gum tissue cannot be expected in these cases, the arresting of progress of the disease is all that can be accomplished, the object of ionization being to overcome infection by pathogenic microorganisms, which have established themselves in the weakened tissues. The

chronic aspect of the disease affords the opportunity to nature's barrier to prevent ingress of organisms into the deep structure of alveolus; the slow inflammatory process stimulates deposition of bone salts and builds up a thickened nodular structure of osseous tissue about the roots of the teeth, eliminating the rarefying osteitis present in acute forms of the disease, so that the bone which has been attacked on that surface in direct contact with infection from without is dense and



FIG. 23.—Chronic periodontal disease.

capable of rigid support of teeth which have lost one-third or even a half of their alveolar border. Fig. 23, a radiograph of chronic periodontal disease, shows thickened heavy bone about the roots with extra heavy linea dura and strong bone forming the floor of the interspaces. Compare this with (Fig. 24) a radiograph of acute periodontal disease. The rarefied bone with loss of linea dura is very marked.

This form of periodontoclasia frequently attacks otherwise perfect dentures, and in undertaking the treatment ioniza-

tion should not be relied on altogether. It is useful in dealing with the infection of the tissues, but great care should be exercised in discovering the other factors responsible for the condition. One of these (the most constant) is failure to carry out proper daily hygienic methods of cleaning, despite praiseworthy efforts, often the teeth are overbrushed on the outer surfaces, while the palatal and lingual aspects are neglected, and the interspaces never touched. The daily



FIG. 24.—Acute periodontal disease.

cleansing of the interspaces with silk should be insisted on and a suitable antiseptic wash to be used on the brush constitutes a part of the after-management of the case, which is incumbent on the patient. During the course of treatment should the patient not comply with instructions, and should sticky particles of food still be found adhering anywhere, attention should be called to it and the importance of keeping it away dilated upon. The presence of mouth protozoa in abundance would suggest the prescribing *vin. ipecac.*, a few

drops on the wet brush to be used once a day for a few months, alternating this with a benzoic acid and thymol wash, also to be used on the brush once a day. Every other etiological factor having been carefully eliminated, and the gingival trough restored to normal, it will be safe to conclude that the progress of the disease has been checked, and the case should be dismissed for three months, when it should be seen and close scrutiny of the condition of the gingival trough made. The future management of the case must depend on the condition which now presents, if the tissues have been able to resist further infection (which may be determined by the absence of bleeding on passing an instrument under the gingival border) the success of the treatment may be anticipated, and the case dismissed for six months, after which a thorough clean up and a single treatment by ionic medication twice a year will suffice to keep the denture from further infection.

4. Acute Septic Infection of Gums and Periodontal Membrane without Visible Pus.—In treating this phase of periodontal disease it will be well to bear in mind the local etiological peculiarities which are accountable, and pathological course which any tissue of the body would take, were an irritant thrust into it and kept there, especially if that tissue were bathed in a septic fluid.

The disease is marked by congestion of the gums, either generally or in isolated areas, the papillæ are enlarged and heaped up between the teeth in loose tags, which bleed freely when touched, and the gingival trough is deepened by the loss of dental ligament. This stage is a continuation of the second stage described, and is marked by greater stagnation areas and more accumulation of foreign substances or some irritant cause of longer duration.

Treatment should be based on accurate conception of the etiological factors responsible. The teeth should be examined carefully, and the cause determined. In young subjects it is often associated with mouth-breathing, adenoids, imperfect masticating functions, use of soft pappy foods or neglect of proper hygienic methods. In adults the principle cause is irritants in the gingival trough—calculus, food débris, some mechanical irritant, such as ill-fitting crowns, imperfect marginal edge of a filling, pressure from a denture, extraction of premolars or first molars for regulating, creating spaces, etc. Whatever the cause it should be the aim to remove it. Careful instrumentation and polishing of the root surfaces is always necessary. Copious bleeding usually results at first, but subsides, and swelling decreases after the first instrumentation. In case of sloughing of the marginal edges of interdental papillæ, which creates a painful condition, ionization with 2 per cent. silver nitrate and a weak current relieves this condition.

Electro-sterilization of the gingival trough should be carried out at intervals of every three days, when further instrumentation and polishing are also necessary. A zinc electrode wound with a few shreds of cotton-wool and saturated with 3 per cent. zinc chloride should be used to slowly wipe out the gingival trough with 2 or 3 m.a. current, keeping the electrode in good contact with the tissues while moving it, turning off the current when necessary to replenish fresh wool and more solution when bleeding from the tissues interferes; two or three such treatments with zinc, at intervals specified, followed by two with iodine, will often be sufficient to produce healthy reaction of the gums.

As soon as the use of a stiff brush is possible the patient should be instructed in a useful hygienic method with den-

tifrice and an antiseptic lotion on the brush. Ionic medication should be repeated at intervals until every trace of inflammation has disappeared, which will not occur if any irritant is left in the gingival trough or in contact with the gingival border. Patients should be warned that this condition of gingivitis is the forerunner of pyorrhœa alveolaris, and is liable to speedy recurrence if strict attention is not paid to daily hygiene. This stage of periodontal disease offers great scope for prevention of pyorrhœa alveolaris; patients coming under observation frequently exhibit essential factors leading to more serious stages of disease, which if recognized and removed forestalls its ultimate inevitable development. The existence of loss of balance in the articulation, particularly in young subjects, where, for example, the loss of a first molar not only results in elongation of the opposing tooth, but the creation of spaces at the adjoining teeth by their tilting toward the vacant space, and the creation of undue stress on certain teeth called upon to undertake extra work. Judicious replacing of such a loss might in itself alone prevent the development of pyorrhœa in a wide area of the denture. The loss of several molars, either maxillary or mandibular, which are not replaced by artificial substitutes, invariably creates undue stress on the anterior teeth, which in time produces the worst form of rarefying osteitis and pyorrhœa. Superior protrusion and postnormal occlusion in adults often result in the lower incisors occluding on the gums, providing an irritant cause of periodontal disease. The grinding of the surfaces of the lower incisors relieves this and prevents the development of disease at this site. In a word, every existing irritant to gums or roots of teeth should be recognized and treated in the early stages with a view to establishing a normal and functional denture.

5. **Chronic Periodontal Disease, with Pus.** "**Pyorrhœa Alveolaris.**"—The treatment of pyorrhœa alveolaris, like other preceding stages of periodontal disease, should be based on a clear conception of the various etiological factors. So varied are these, and often so subtle, that the greatest difficulty is presented in detecting the real exciting cause. Detailed description of the etiological factors has been chronicled in another part of this work, and may be summed up under two headings—irritants and microörganisms.



FIG. 25.—Disease due to subgingival calculus alone.

The clinical aspect of cases of pyorrhœa is usually convincing at a glance, but radiographs of the condition of the alveolus should be procured to determine the extent of rarefaction of the bone, as well as to reveal such exciting causes as faulty fillings or crowns, and also the depth of pockets. Much useful diagnostic information can be deduced from the study of radiographs, which often serve the purpose of determining at once the degree of virulence of the disease, and the possibility of dealing successfully with it. As a rule the encroachment of subgingival calculus alone causes destruction of the marginal edge of alveolus, and leaves an abnormally blunt appearance of the bone between the teeth, the linea dura being well defined.

In cases of distortion of normal occlusion, rarefying osteitis is usually more general with loss of the linea dura and more tapering or irregular bone between the teeth.



FIG. 26.—Due to abnormal occlusion.

The complete destruction of periodontal attachment (Fig. 27) would indicate prompt extraction or the destruction of alveolus beyond the level of the bifurcation of mutilated teeth (Fig. 28) would furnish the conviction that treatment in such circumstances can be but palliative. When



FIG. 27.—Complete destruction of bone.

systemic derangements of various kinds are attributable to toxins from oral sepsis, the radiographic appearance of the alveolar bone is somewhat a guide. The presence of pus with

marked rarefying osteitis, loss of the linea dura, decided enlargement of sockets and existence of deep pockets lend credence to this suspicion, but are not essential, for severe constitutional disturbances are sometimes caused when

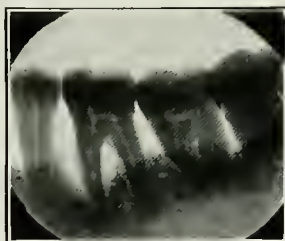


FIG. 28.—Exposed bifurcation.

slight periodontal disease only is present, and the radiographic appearance of the bone reveals but slight changes. Radiographic evidence is sometimes unreliable in diagnosing severity of cases, and does not coincide with the clinical

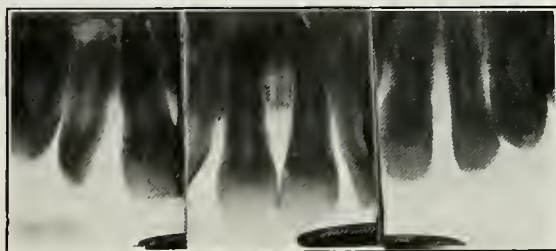


FIG. 29.—Extensive rarefying osteitis.

aspect; if deep pockets exist on either labial or palatal surfaces, not extending to the interspaces, these do not show on the picture, the shadow of the roots obscuring what may be a very serious condition.

A general description of treatment of pyorrhœa, as a whole, does not embrace salient points in a comprehensive form to the student desirous of information on the complex problems which confront him in practice. Every case which presents itself should be first carefully examined and the local etiological factors present ascertained before commencing treatment, with a view to removing the cause and applying the means of restoring diseased tissues to normal.

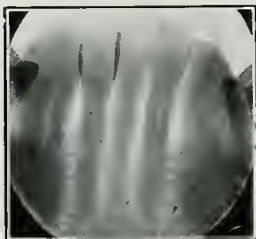


FIG. 30.—Affected lower incisors.

I. The class of cases (the simplest) where the lower incisors are loose with pus exuding on pressure and a state of periodontal disease extending from this area backward in a less degree with no visible pus elsewhere. The cause is generally salivary calculus on the surfaces of the teeth, and subgingival calculus the product of inflammation. If on occlusion these teeth move beyond their normal limits of motion (which can be ascertained by placing the finger lightly on the teeth and instructing the patient to close sharply) the occlusal surfaces should be ground until complete relief from this traumatic factor is obtained. The removal of all foreign matter from the teeth surfaces with instruments and perfect root surgery, which consists not only in removing every particle of subgingival calculus but a slight planing of the

denuded surface of cementum which has been pus-soaked for a lengthy period, leaves it absolutely smooth. Instrumentation should be carried out farther back to every tooth on which the slightest nodule of calculus adheres, with a view to arresting the disease. Next polishing the surfaces of the teeth with powdered pumice made into a paste with aromatic sulphuric acid and water carried on engine brushes of wheel or cone shapes; the interspaces require most careful attention also, which can best be done by passing fine lava polishing strips between, stretching them taut and firmly rubbing the gritty material against these surfaces. These strips of narrow width work effectively on the surfaces of all teeth under the gums by encircling a tooth and bringing both ends on the outside, then, with a reciprocal pulling motion the tape works itself under the gum margin and polishes those surfaces most effectively. During the whole course of treatment at every sitting the teeth should be polished to the highest state of perfection.

The root surgery and polishing process having been thoroughly carried out, the loosened teeth should be ligatured with fine gilt wire (Angle's fine), beginning at the nearest firm tooth (usually the cuspid), the wire should be interlaced and stretched taut just above the bulging contour of the teeth to prevent slipping down on the gums and ending the wire on the opposite side on a firm tooth (Fig. 31). The twisted ending should be tucked away to prevent any irritation of the soft tissues. This ligaturing should leave the loosened teeth perfectly rigid, immovable in the enlarged sockets, in which position they should be held to promote physiological rest until regeneration of bone takes the place of the mechanical device.

The possibility of regeneration of bone once affected by

rarefying osteitis has been freely denied by some authorities, but clinical and x-ray evidence is convincing that this actually occurs. Dr. T. Sydney Smith¹, of California,



FIG. 31



FIG. 32.—*a*, radiograph taken April 27, 1914, showing destruction of the alveolar process; *b*, radiograph of the same taken August 15, 1914; *c*, the same taken December 7, 1914, showing regeneration of bone.

pointed this out at the Sixth International Dental Congress, and subsequently furnished me with the accompanying example, Fig. 32. It is necessary in order for this to take

¹ Transactions of Sixth International Dental Congress, p. 213.

place that the teeth be kept steady in a condition of physiological rest.

Root surgery alone is insufficient; the tissues and alveolar bone have been so weakened by chronic inflammation and the presence of pus that pathogenic microorganisms inhibit them and are able to flourish. Irrigating or syringing the pockets is ineffective, because antiseptic lotions are not long enough in contact with tissues into which organisms have penetrated to have any effect on them, and regeneration of tissue cannot take place in the presence of opposing pathogenic organisms.

The author has therefore instituted ionic medication to cope with this etiological factor. At each sitting after the first surgical operation, which is usually attended with copious bleeding, the gingival trough should be ionized with zinc ions; a zinc electrode wound at the point with a little cotton-wool saturated with 3 per cent. zinc chloride or sulphate should be passed into the gingival trough to the bottom of the pockets, the patient holding the indifferent electrode. Current should be gradually turned on from the generator, until it is felt. Ionization should be carried out with the object of obtaining deep penetration with a sufficient dose of ions to affect the deep-seated organisms. In this respect the dose must be judged in direct ratio of current strength to time employed, that is, if the patient will only tolerate two milliampères the electrode should be held in position for half a minute to ionize a pocket between two incisors, and should be gradually moved around the neck of the tooth into another pocket between adjacent teeth, but if 3 or 4 m.a. are available the time required for proper penetration of ions will be proportionately reduced to one-third or one-quarter of a minute.

Care should be taken that the entire gingival trough is subjected to the direct contact of the electrode, which is done in its slow movement about the neck of the tooth, the pocket being flooded with the zinc solution, ionization continues at some distance from the actual contact of the electrode, although penetration is deeper where contact is made.

The operation should be repeated every second day until pus has disappeared and inflammation has subsided, when the interval can be extended to three days, and later to once a week, when ionization with iodine, using the negative pole, should be substituted for the zinc. A good deal of judgment is required in ascertaining when the tissues have had sufficient treatment. Restoration to healthy appearance is not in itself a sufficient guide. Existing conditions, such as a delicate osseous frame with fragile alveolar bone, or the reverse, the former imposing longer care and attention than the latter, also the general health, must be considered; when general constitutional disturbances exist the tissues repair more slowly than when local affections only exist. The current strength available also affects the dose of ions. If only 1 m.a. has been used the restoring effect will be much slower than if 3 or 4 m.a. have been possible. One condition, however, always imposes itself in all cases, and that is it must be the object in view to so far restore the gingivitis to normal that in the act of eating foodstuffs will not readily pass into the gingival trough. The gums should be tough or contracted about the necks of the teeth before the patient can reasonably be expected to effectively continue daily treatment on hygienic lines.

Nor does the operator's responsibility rest here, even in the most perfect looking result. Few people presenting a state

of advanced pyorrhœa possess any real knowledge of how to keep their teeth. Their methods have failed, and it is the duty of the operator to discover the lines on which they have worked, to correct their faults and train them in proper methods. Tooth-brushes, as a rule, are useless things. One may examine hundreds on sale and find only a small percentage suitable for the purpose intended. A stiff brush shaped somewhat like Fig. 33, answers the purpose well, and it should be explained to patients that not only should the teeth be brushed, but gum massage with the brush is essential



FIG. 33

to tone up and keep gum margins in a condition capable of resisting reinfection. A good dentifrice should be used to keep the teeth polished, followed by a few drops of some antiseptic wash on the brush, such as:

R—Thymol	grs. iij
Benzoic acid	ʒss
Ol. cinnamon	℥ x
Acid carbolic	℥ xxx
Otto rosæ	℥xv
Alcohol	ad. ʒiv—M.

Interspaces should be cleaned at least once a day with waxed floss silk. In cases in which loosened teeth are ligatured or permanent splints support them, individual cleaning

of spaces must be done. A bit of orangewood thinly pointed, moistened with antiseptic and dipped into powder, should be used to polish these surfaces, which neither brush nor silk can get at, care being taken to rinse away all powder after using it.

Details of postoperative treatment of pyorrhœa cannot be too carefully instilled in the patient's mind, and constitute after-management of cases, on which depends the success or failure in the treatment of pyorrhœa. After a course of treatment the case should be dismissed for periods varying, according to its severity and the ability of the patient to carry out instructions, from six weeks to three months, when a thorough examination should be instituted to determine if instructions have been carried out and if faulty operating has left the smallest particle of irritant in the gingival trough, causing reinfection and a state of slight inflammation at that point. According to the ability of the patient to manage his own case, the future intervals when the case should be seen must be determined, usually three times or twice a year. No cure of pyorrhœa can be ascertained in less than a year after treatment, but two or three years are better, some cases requiring constant care for even longer periods, but with coöperation of the patient and a determination to overcome every etiological factor it is remarkable the proportion of cases which respond to treatment, the teeth remaining functional and free from sepsis.

II. The class of cases in which the superior incisors are principally attacked on the palatal surfaces, pus pockets extending some distance on those surfaces, and to the approximating spaces until the labial surfaces (which are probably well brushed) are reached and to all appearances are healthy. The teeth are often abnormally spaced, due to slight protru-

sion. The cause is generally neglect to cleanse those surfaces, and here subgingival calculus alone is found.

Etiological factors here are usually neglect or impinging of the lower incisors from faulty occlusion or faulty occlusion and stress brought to bear by the loss of molars, or even premolars (Fig. 34), by which undue strain is placed on the incisors, establishing rarefying osteitis and chronic inflammation of the periodontal membrane. Occasionally the lateral



FIG. 34.—Extraction of first premolar the cause of rarefying osteitis about the lateral incisor by faulty articulation with inferior cuspid.

incisors alone are affected by faulty occlusion, the lower cuspids striking and pressing them outward at every closure of the jaw.

Treatment consists in thorough instrumentation and polishing, the relieving of abnormal stress by grinding the opposing lower teeth, replacing into normal position teeth which have been forced out and thorough ionization of the gingival trough. Instrumentation should include the labial surfaces

even if apparently healthy, here a granular layer of subgingival calculus invariably exists. Replacing of abnormally placed teeth may involve considerable mechanical ingenuity, not only in regaining normal positions, but, what is more important, in retaining them there. Fig. 35 shows an extreme case of protrusion, caused by occlusion of the lower incisors on the roots of the uppers, and also the loss of various teeth in the maxilla and mandible. In this case



FIG. 35.—A pronounced case of protrusion and pyorrhoea in an adult.

Angle's retraction head-gear was employed to obtain the position shown in Fig. 36; grinding of the surfaces of the lower incisor was also necessary; lost teeth were replaced by artificial substitutes on plates, thus relieving pressure on the anterior teeth. An upper retaining plate was also made to be worn at night, with a wire arch attached, which fitted closely to the surfaces of the superior incisors, preventing the possibility of moving out of position. In less affected cases, where teeth have moved slightly and pockets are not deep,

ligaturing for a time after relieving pressure is all that is necessary until new bone is formed about the enlarged socket. The attachment of permanent splints is indicated in cases where malocclusion is not accountable for incisors becoming malplaced, but when long-standing pyorrhœa has brought about rarefying osteitis and loss of periodontal attachment principally in the palatal aspect, such teeth require permanent support after replacement. The obliteration of the pockets

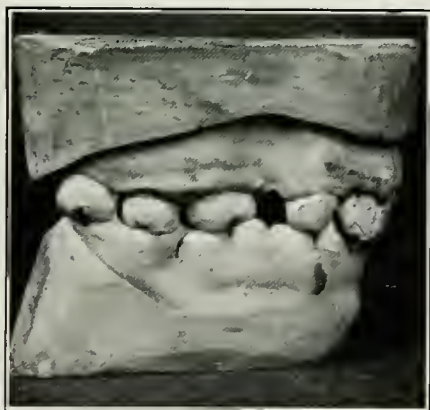


FIG. 36.—The position after treatment.

by retraction of the teeth is in itself a means of checking the disease if instrumentation has been successful; but the tendency is to move outward again. Splints can be attached to live teeth without destruction of the pulps, and if one or two pulpless teeth exist they should be utilized to obtain more durable attachment. In the case of four vital incisors, fine drill holes in parallel midway between the pulp and surface margin of each tooth, slightly larger than the platinum pin of a plate tooth, 5 mm. deep, constitute the plan on which

to construct a splint, which can either be cast or constructed by soldering iridioplatinum wire attachments on platinum foil burnished on the impression containing the pins *in situ*.



FIG. 37.—Splint which retained these teeth for sixteen years.

The ends of a splint for this purpose should extend to and rest on adjoining firm teeth on either side, not necessarily to be attached to them. Fig. 37 is a radiograph taken just before removing the teeth which were retained for sixteen

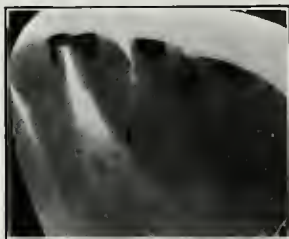


FIG. 38.—Splint on incisors.

years by the splint. In the case of two devitalized laterals, which have moved outward the splint can be constructed on them with long pins in the pulp chambers, the bar resting

on firm centrals. Fig. 38 shows a splint on incisors which retained these teeth for a number of years. When separation of central incisors alone occurs from disease between them, instrumentation, ionization and replacing and splinting them (Fig. 39), is very effective in curing and arresting further spread of periodontal disease to adjoining teeth. Splints on the superior incisors give little trouble to keep clean, and the fear of caries, as a result, can be ignored even in only ordinarily careful patients; individual cleansing of the spaces with wood points by the patient and semi-yearly (at least) polishing by operator is sufficient.



FIG. 39

III. The class of cases in which the disease starts in the molar region, affecting the premolars less, and the incisors possibly very slightly, or not at all. Pockets are usually established between the teeth and posterior to the last molars, where a flap of gum retains septic matter. The palatal roots of the first and second molars are often denuded of bone and tissue covering. Septic infection is generally worse between the teeth, where neglect of hygiene has probably been the principal cause of disease. Subgingival calculus

of a hard, tenacious character usually encircles the roots of the teeth impinging on the remaining periodontal attachment. Instrumentation in this position is very difficult and imposes on the operator a high degree of skill, patience and exactness (upon which hangs success) and failure often stares one in the face, in spite of most diligent and honest efforts to overcome this obstacle. This irritant cause (subgingival calculus) must be completely removed, a tedious operation for patient and operator alike, which may entail repeated searchings under the gums for small particles, and also the slight planing of the root surfaces. Ionization with zinc ions should be carried out, using the strongest current strength available with comfort for the patient; 5 m.a. is nearly always possible, and some patients can stand 10 or even 15 m.a. current.

The period of treatment should extend at intervals of every second day, until not only pus has disappeared and the tissues appear healthy, but the gum tissue has shrunk close to the roots of the teeth, and a solid tough floor of tissue formed between the teeth, which can resist ingress of food particles to the gingival trough.

The polishing of interspaces should be effected with lava strips, and all enamel surfaces polished to the highest degree of smoothness with port polishers, brushes or buff wheels and a gritty acidulated paste. The articulation of the teeth requires most careful study to ascertain if traumatic occlusion exists; abnormal limits of motion on closure should be detected; often there is no difficulty in discerning this; a single molar or pair of molars may be quite loose, and reveal considerable movement on occlusion. The radiograph also will show considerable rarefying osteitis with loss of linea dura. To correct traumatic occlusion, judicious grinding

of the planes of occlusion should be done with carborundum stones, after marking the contact points with thin carbon paper, repeating the process of biting on the paper and grinding until undue stress is relieved and proper balance established in the denture.

An illustration of the effects of traumatic occlusion on a denture is well shown in the accompanying radiographs and photographs of the denture. Fig. 40 shows abnormal



FIG. 40.—Abnormal occlusion due to extraction of premolar.

occlusion in a patient, aged forty years, due to the pernicious operation of extracting a pair of premolars to correct irregularities in childhood. This produced impinging of the inferior cuspids on the superior laterals and abnormal articulation in the molar region, with the result that rarefying osteitis was specially marked on the palatal surface over the laterals and second molars (Fig. 41) and abnormal movement of these teeth, with a spreading attack of periodontal disease over the

entire maxillary denture. Fig. 42 shows radiographs of the condition of the bone. Treatment consisted in relieving

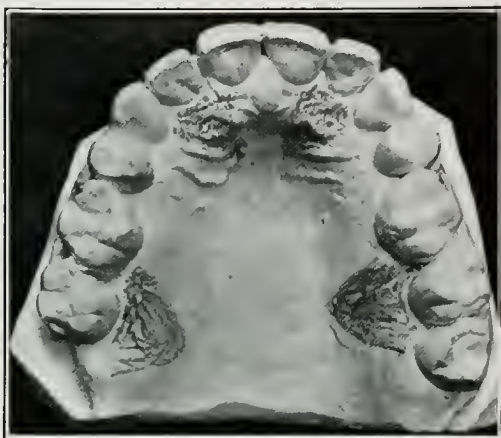


FIG. 41.—Rarefying osteitis in region of laterals and molars.

undue pressure by grinding the lower cuspids and ligaturing the superior laterals for three months and relieving strain

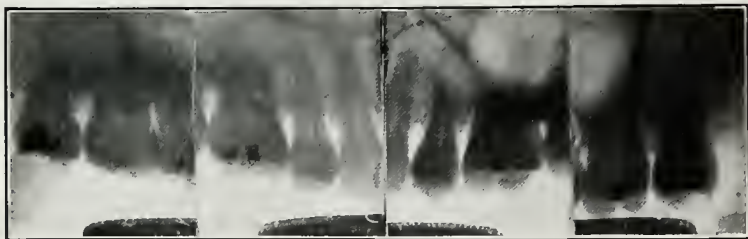


FIG. 42.—Radiographs of the maxillary denture.

on the second molars. Subgingival calculus of a hard black kind was removed and the gingival trough ionized with zinc

and iodine ions. The case treated in March, 1913, has been seen once a year since and is now in perfect condition; the loosened teeth are as firm as the other teeth, and the disease in general arrested. The patient volunteered the statement that her mother, now edentulous from pyorrhœa, was treated by extractions like herself, and first showed the same symptoms at precisely her age (forty), which convinced her that something was going wrong with her teeth.

Postoperative management is of vital importance. The patient's habits in relation to daily hygiene should be inquired into and faults corrected. Dietetic errors, due to modern methods of food preparation, cannot be changed in a day, but advice can be given to counteract some of its effects. The adhesion of soft, sticky food to the approximating surfaces of teeth in the molar region is one of the most difficult aspects of recurring infection in this part, but this can be often overcome by advising (in addition to hygienic methods already referred to) the use by patients of fresh fruit after the last meal of the day. This is an effective means also of preventing the formation of salivary calculus in other parts of the denture. Environmental forces should also be considered and inquired into. The effect of closed windows, unhealthy atmospheres, sedentary life, lack of exercise—everything in the habits of the patient opposed to natural and primitive environment comes under the category of investigations the dentist should institute for the betterment of chronic disease of the alveolus.

IV. The class of cases differing from the foregoing (so frequently seen in this country), caused by the ruthless extraction of molars, sometimes maxillary unilateral, sometimes bilateral, the mandibular molars being retained, or *vice versa*, the mandibular, the maxillary retained. The

undue stress of mastication thus brought to bear on the anterior teeth is the undoubted cause of periodontal disease developing in the denture, which therapeutic and surgical treatment alone cannot overcome. Dentures are often supplied, which are a source of irritation, or inadequate in restoring balance. Treatment in such instances consists in the already described surgical and ionization methods, and in addition careful restoration of balance of the denture by replacing lost teeth by methods which ensure the most perfect articulation in the molar region without producing irritating pressure on the gingivus in the vicinity of diseased teeth. Plates should be constructed with well-fitting saddles to resist the strain of mastication, and if clasps are necessary they should fit clear of the gingival border without a dragging influence on the teeth clasped. In the mandible it is often possible to construct plates carrying lost molars, on the principle advocated by Mr. Badcock, by a solid wire bar fitted to the curve of the jaw below the gum margin, attached to well-fitting saddles and clasps on the premolars.

The most perfect method of restoring balance of articulation, and relieving undue stress by loss of molars or premolar teeth, when circumstances permit, is by fixed or removable bridges. These should be constructed so as not to violate the principles of antiseptic surgery—a difficult matter, which, by many, will be stigmatized at once as impossible, but which nevertheless can be carried out by those expert in this method of restoration. One of the most antiseptic methods of bridging in these cases consists in the “suspension” bridge (Fig. 43), which can often be constructed on inlay abutments. This leaves a clear space of considerable width between the restored surface and the gums, and patients should be instructed in the method of cleansing by occasionally passing

a strip of tape underneath and polishing the surface which cannot be cleansed by the brush.

V. The class of cases which owe their origin to badly constructed fillings or crown and bridge-work is exceedingly common, and is confined to mouths prone to caries, and consequently not so predisposed to virulent periodontal disease. The disease is generally confined to local areas, influenced by this exciting cause, but may have spread generally by contagion before discovered. Treatment consists in removing the cause and carrying out the principles of surgical and



FIG. 43.—Suspension bridge in position.

therapeutic treatment by ionization. Pockets established between the teeth which have been filled leaving imperfect edges or with imperfect contour, usually assume a wedge shape (Fig. 44), the lodgment of food by pressure forcing the gum against the absorbing alveolar bone. The regeneration of bone and gum in this circumstance does not occur and an awkward space exists, which requires special attention by the patient. Fillings which are the cause of such a condition should be removed and the disease treated by ionization until the tissues have resumed the normal and a tough, well-contracted gingival floor is established, when they should

be replaced with fillings having anatomically correct contact points and perfect cervical edges. Flat, shapeless fillings simply aggravate the trouble by providing spaces which permit of the lodgment of food. When these spaces occur fairly near the front of the mouth, in addition to cleansing with silk, some patients have used an adaptable steel instrument which, on several occasions, the writer has entrusted to them with results which were most gratifying.



FIG. 44.—Wedge shaped pocket.

As regards crown and bridge-work, there is no doubt that a vast amount of oral sepsis is created and maintained by faulty construction or unwise selection of cases for this much abused method of restoration. But of the two evils—a questionable amount of sepsis from a well-constructed crown or bridge, or the loss of balance in the denture from the lack of teeth—the former will appeal to the minds of all who understand the hopelessness of undertaking to cure even slight periodontal disease, not to mention established pyorrhœa. Crowns and bridges are often so constructed that they can only be classed in the category of “septic surgery,” so justly condemned by Hunter; these should be removed, and when attached to roots hoplessly affected by pyorrhœa, the roots should also be removed: the restoration of the

denture to health should take the lines of treatment already described under instrumentation, polishing, ionization and proper hygienic methods.

The question of replacing crowns or bridges after the tissues have been restored must be governed by circumstances. Gold shell or banded crowns on teeth which have been affected by pyorrhœa from no other cause than an ill-fitting band in contact with the gingivus, if replaced by even most perfect work might be the cause of recurrence; but porcelain crowns and bandless crowns of all descriptions, which have



FIG. 45.—Crown with a perfect joint.

no joints to catch and retain foreign matter (Fig. 45), have no more influence on periodontal disease than natural teeth affected similarly, and can be retained in quite as healthy a condition.

VI. The class of cases which some writers would attribute to “predisposing causes” furnish the most difficult to deal with. They are often associated with a frail osseous frame, thin, narrow jaws, a delicate constitution or the accompaniment of some systemic derangement, which, in the majority of cases, is caused by oral sepsis.

The treatment of periodontal disease for patients in this

category imposes on the dentist a grave responsibility. Cases are usually referred to him by the medical practitioner, who is quite incompetent, as a rule, to discern for himself if oral sepsis in reality exists, and is likely to be the cause of infection accountable for such ill effects as enumerated by Hunter, viz., "the general ill health, dirty, sallow complexion, the indigestions, the gastric and intestinal troubles, the anemias which resist treatment, the tonsillitic, pharyngeal and glandular troubles of children, the chronic rheumatisms, obscure fevers and blood poisonings, etc." Here judgment must be exercised in determining, apart from the existence of septic infection of the mouth, the degree of infection and the possibility of retaining the infected teeth. Under the cloak of "predisposing causes" a section of the profession endeavor to hide their own inability to overcome the increased difficulties caused by relationship to other existing diseases, they ignore the possibility of local infection being the sole cause of constitutional disorders, and that with the disappearance of local septic infection the constitutional derangement due to toxins will disappear, and the dreaded intractable disease resolve into an ordinary local infection amenable to local treatment, and the regeneration of alveolar bone and tissues take the ordinary course, aided by improved general conditions.

Under the heading of "Cases not Favorable for Treatment," Colyer¹ includes "those showing well-marked signs of rarefying osteitis and with general and local conditions, which indicate that the tissues have little recuperative power." In other words (as is carefully explained in the chapter under this heading), every tooth with a pyorrhœa

¹ Chronic General Periodontitis, p. 88.

pocket actively discharging pus should be extracted "at the earliest opportunity." Any other line of treatment, in his opinion, is unsatisfactory, and he urges that "early removal of the teeth in cases in which the disease is making progress is also of practical importance." This doctrine of free extraction, that is extraction of all the teeth, coming from such a well-known authority, who has done useful work on



FIG. 46

morbid anatomy and pathology of periodontal disease, one might hesitate to condemn had not Colyer, in his argument for this course, condemned it himself by publishing skia-grams¹ (Fig. 46), reproduced here, of a typical case which shows conclusively that proper local treatment (which

¹ Chronic General Periodontitis, p. 90.

extended over eighteen months) had not been carried out. Examine the radiograph—more beautiful examples of subgingival calculus which existed in the first picture and had increased in the second, could hardly be found! The calculus had not been removed; the irritant cause of pyorrhœa had been left behind during all this time. The kind of local treatment employed is not chronicled, but whatever it was it did not include removal of subgingival calculus, and on the strength of the results, which were bad, the patient was made edentulous. Vaccines, which were used, could have no effect while the local cause remained. That should not be the lines on which the dental profession should work. Some serious effort should be made to retain the denture in these cases, for it will be found that often the disease can be checked and the septic source eliminated, it may be only temporarily at first, but carefully following up the treatment and watching over the case to prevent recurrence of the slightest inflammation of the gingivus, eliminates sepsis of the oral tissues, which, if it is the source of toxins, will have the desired effect on the associated pathological condition elsewhere and in time be reflected on the local condition. The writer has in this way saved many a denture, where the constitutional disorders have been shown to react, as in one case of anemia particularly, which took three years to completely disappear, but eventually it did, and the local affection also, leaving the patient with a functional denture, which in appearance and utility far surpasses the most skilfully constructed artificial substitute.

Advanced cases of pyorrhœa, when no active pathological lesion is suspected, but a frail osseous frame or delicate constitution indicates the likelihood of ready absorption of organisms into the medulla of bone or the weakening of the

gastric defences, provides a difficult class of subjects to deal with. Nevertheless, if rarefying osteitis has not advanced generally so as to loosen all the teeth, but only in certain areas, it is of great advantage to these people to retain all teeth possible to provide organs of mastication and support for artificial substitutes. Thorough instrumentation and electro-sterilization will usually clear up the disease, but a great tendency at first to recurrence is generally observed. This tendency should be mentioned to the patient and instructions given to return three months after treatment for a clean-up and ionic medication of the gingival trough. Future management of the case rests principally with the patient, who should be well instructed in an effective method of hygiene and gum massage with the brush. These cases require to be seen at regular intervals of four months for a year or two, after which it is often gratifying to find improved tone in the tissues, which resist infection and a decided strengthening of the alveolar investment.

The tendency to recurrence need not be interpreted as failure, especially if the sockets are not enlarged and teeth consequently loosened; but the possibility of some irritant cause being left behind, or tissue not having sufficiently recuperated to resist microorganisms, should be etiological factors kept in mind, and treatment continued by additional instrumentation, including the planing of the root surfaces where pus persists and prolonged electro-sterilization. The presence of amœbæ can invariably be detected, and the author has found that the use of tincture of ipecac on the wet brush, once daily in the morning, as well as the antiseptic wash at night, assists in overcoming this tendency to recurrence of the disease.

It has been the writer's gratifying experience to have on

record many cases of this class, which resisted treatment at first for two or even three years, then became absolute cures. In order to wait so long for results, serious pathological symptoms in other parts of the body must be absent.

VII. Cases associated with active pathological lesions elsewhere are exceedingly common. In the author's experience they include the following in order of greatest frequency: General ill health, rheumatic symptoms, gastric and intestinal troubles, tonsillitis, obscure temperatures, anemia, rheumatoid arthritis, glandular swellings, neuras-

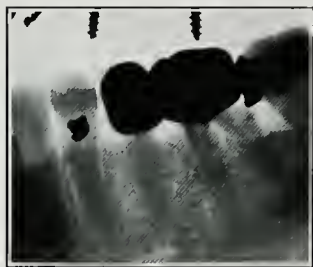


FIG. 47.—Space liable to reinfection.

thenia, inflammatory diseases of the eye, etc. Treatment of pyorrhœa in this class of cases imposes on the dentist scrupulous care that not only is the source of sepsis removed, but that the mouth is left in such a hygienic condition that it is humanly possible for the patient to keep it so. Potential sources of infection, which cannot be reached in the daily hygienic efforts of the patient, should be eliminated by extraction, such as multirooted teeth with exposed bifurcations denuded of alveolar and gum covering (Fig. 47); deep pockets in the molar interspaces (Fig. 48); semi-impacted mandibular third molars; teeth with pyorrhœa abscesses,

especially in the mandible, where gravitation of pus usually extends rapidly toward the apices; teeth so loosened by alveo-

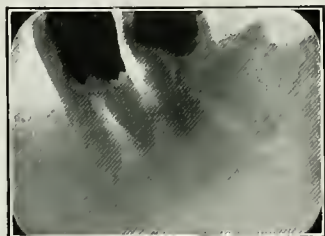


FIG. 48.—Deep pocket between molars.



FIG. 49.—Pyorrhoëa abscess.



FIG. 50.—Granuloma.

lar absorption and rarefying osteitis that abnormal movement takes place on closure of the jaws or in mastication; and all dead teeth with granulomas (Fig. 50) or chronic abscesses

discharging on the gums, indicated by the radiographs. These furnish instances when extraction of teeth becomes necessary, as an adjunct in the relieving of pathological general symptoms in treatment of the disease by conservative methods, details of which have already been described, consisting of instrumentation, cleaning and polishing of tooth surfaces, removing of irritant factors such as faulty crown and bridge-work, imperfect fillings, restoration of normal balance in the articulation and dealing with the micro-organisms by electro-sterilization of the gingival trough about all teeth retained.

A complete cessation of the disease must be ensured, no pus-yielding pockets must be allowed to persist and the gums should contract about treated teeth, so as to resist reëntry of soft food into the gingival trough.

Periodontal disease which has existed long enough to affect the alveolar bone seriously, with deep pockets about the teeth and denuded sockets, from which badly diseased teeth have been removed, involves the bone in areas of necrosis of varying extent, necessitating surgical interference during treatment. Deep pockets passing below the alveolar level produce a surface of necrotic bone, which should be scraped or curetted to remove all dead bone. This in many cases can be done with a spoon-shaped instrument, or in the case of extraction the socket should be burred with a large round bur until the necrotic surface is removed. The particles of detached bone should be removed by syringing with an anti-septic lotion or by wiping out the pockets with medicated cotton-wool on a barbed probe. The persistence of pus at a particular location is usually an indication that necrotic bone remains and further curetting is necessary. The use of a local anæsthetic is often required in the operation on necrotic

areas; the tissues are readily anæsthetized, which facilitates thorough work.

When oral sepsis is the source and supply of toxins responsible for other pathological lesions present, successful treatment invariably relieves it, and often within a very short time signs of improvement are manifest. With improvement in the general symptoms the local inflammation takes on more active improvement also, both depending on removal of sepsis. It has become very general for medical and dental practitioners to advise extraction of every tooth in the mouth in cases in which marked constitutional symptoms are attributable to oral sepsis. This drastic procedure invariably involves numbers of useful sound teeth, which can readily be retained without the least risk to the patient if proper dental treatment is carried out, and the writer has no hesitancy in condemning it in most emphatic terms. The following is an example of how unnecessary this method often is.

Miss R., aged twenty-nine years, consulted me in January, 1898, advised by her doctor and dentist to have all her teeth extracted. Pyorrhœa pockets existed on the palatal aspect of the superior incisors, extending nearly to the apices, with considerable discharge of pus and teeth much loosened. The lower teeth were all slightly affected. The patient was nervous and debilitated, the effects of alimentary toxemia.

Treatment consisted in extracting one tooth (a premolar) and reducing every etiological factor responsible by methods of instrumentation, retraction of superior incisors (see Fig. 51), electro-sterilization and restoration of balance in the denture by artificial substitutes. In eight weeks the pyorrhœa had completely disappeared and the general health improved to a remarkable extent. For twenty years the patient has retained her teeth in a functional and healthy condition and

enjoyed good health, in the meantime requiring no more than the ordinary attention, which all patients should have.



FIG. 51.—The upper model taken about ten years after treatment shows growth of alveolar over incisors.

General Considerations.—Instrumentation and the polishing process almost invariably produce sensitive surfaces of teeth, which react more or less violently to thermal changes and the action of acids and sweets. This discomfort is often bitterly complained of by patients, and is, indeed, an awkward result, which if it persisted would condemn the method of treatment; but fortunately the obtunding effect of zinc ions is never-failing, and although at first this very symptom retards the use of heavy currents for ionization, still the sensitiveness is usually quickly overcome, and by the end of a course of treatment it generally disappears, or else is so reduced as to be readily tolerated, and eventually it disappears. Occasionally it persists, and when it occurs in positions such as the roots of molars or palatal and lingual surfaces of other teeth, where black discoloration of the

teeth does not matter, it can be immediately relieved by drying the sensitive surface and painting it with saturated solution of nitrate of silver. This, if allowed to stay on for a few minutes, will obtund the sensitive dentinal nerve endings and relieve the condition after one or two such applications. In front teeth ionization of the tooth surface with zinc ions will eventually produce the obtunding effect.

In many cases of pyorrhœa the teeth are coated with transparent or translucent sticky mucus, which is septic. This should be carefully removed by brushing at each treatment with an alcoholic wash or acidulated powder, leaving absolutely polished surfaces. The interspaces should be cleaned with fine strips. Occasionally this mucus secretion is found in large quantities, the mucous glands being infected secrete an abnormal fluid resembling pus. Patients will complain that in the morning the teeth are covered with "pus," when there is no apparent discharge from the gums. This condition can be relieved by ionizing the mucous surfaces with 10 per cent. iodine solution, using a flat platinum electrode covered with cotton-wool saturated with the solution. The gums and mucous membrane above the level of the roots of the teeth should be treated. Usually 10 m.a. current can be used, and the iodine, as it is ionized, passes into the tissues, leaving a bleached appearance to the medicated wool. By slowly moving the electrode over the gums every part should be medicated.

Fœtid odor, which is characteristic of the disease, rarely persists after the disease is under control, but this disagreeable feature occasionally remains, when no apparent cause from periodontal disease warrants it, and examination of the tonsils or antrum is then advisable. The author has often discovered an infected state of the antrum or tonsils,

which may have resulted from oral sepsis, and until this has been attended to by a throat and nose specialist the condition of the mouth remains intractable, with inclination to reinfection, and treatment of pyorrhœa is not effective. The vigorous maintenance of treatment, which at first should be every second day, and polishing the teeth each time, is intended to keep away the formation of bacterial plaques on the teeth, and reduce to a minimum the influence of organisms in an infected mouth, while ionization of the gingival trough deals effectively with organisms in the tissues themselves. At the same time it is necessary to determine if any irritant specks of calculus are left, even after the most perfect instrumentation, and as treatment advances there is no surer sign that tartar has escaped the instruments, than to find here and there a slightly inflamed interdental papilla or a margin of gingivus. There is not the slightest detriment to the periodontal membrane from reëntering the gingival trough to perform the necessary removal of tenacious calculus, even if this reëntry has to be undertaken at every sitting throughout a long course of treatment; when the tissues are perfectly healthy no bleeding will occur from the use of instruments on the roots of teeth which once would bleed on the slightest touch.

Instrumentation.—The technic of instrumentation must rest with each individual operator and description of instruments would be futile; suffice it to say that vast numbers of instruments are exhibited by instrument makers, which are to the writer's mind totally unsuited to carry out what should be the intention of the operation; the object being to remove all necrotic tissue adherent to the roots and calcareous deposits. Instruments should have keen cutting edges, with rigid shafts of finest steel, and of a size that

will not lacerate unnecessarily overlying soft tissues. Just above the deposited subgingival calculus is always an area of vacant space before reaching fibers of periodontal tissue (Fig. 52); into this space if a blunt-ended hook-shaped instrument is passed, a firm, steady pull in the direction of the shaft planes off adherent foreign matter and removes it. The important part is to accomplish this object by the methods best known to the individual. It should be unnecessary to refer to this purely surgical detail, but the writer's experi-

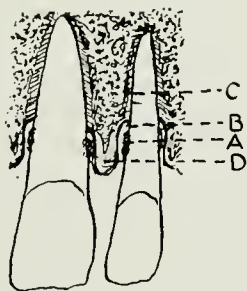


FIG. 52.—A, salivary calculus; B, space above calculus; C, periodontal membrane; D, inflamed gum tissue.

ence is that there is no operation in dentistry so little understood, or so imperfectly performed generally, as the removal of subgingival calculus.

Much can be done toward relieving periodontal disease by skilful instrumentation and after-management of cases alone, but in the author's opinion these are insufficient, as they leave too much to nature in the matter of overcoming infection of the gums, periodontal tissue and alveolar bone, into which pathogenic organisms penetrate and are not reached by any form of antiseptic spray or irrigation.

Other Methods.—There are doubtless many other methods instituted by successful practitioners for dealing with advanced periodontal disease, and claims of curing it are freely made, and doubtless with skilful instrumentation, aided by osmotic methods of applying certain drugs, and with due consideration to every etiological factor, these results are attained, but the writer emphasizes the quicker and more universal results obtained by medicating the tissues with antiseptic ions, which deals with deep-seated organisms at the outset, and relieves the strain imposed on recovering tissues by providing antibodies to combat pathogenic organisms.

Vaccine Therapy.—The method of treatment by vaccines, which is based on one of the most important scientific discoveries in medicine, is irrational and impractical in the case of this localized disease, which is admitted to be dependent on the action of no particular organism, but purely secondary in infection by any of a complex group of organisms ever present in the oral flora. The selection of one or two varieties of organisms from which to prepare an autogenous bacterial vaccine, in the present state of our knowledge, is hazardous, and even if precise knowledge can be obtained by staining organisms in specimens of diseased tissues removed from the patient, vaccine therapy is quite unnecessary in the treatment of this local infection, which can be treated successfully from a local standpoint. In grave constitutional disturbances due to absorption of toxins from a pyorrhœal source, undoubtedly vaccines composed of correct organisms responsible possess distinct value in establishing immunity, but no cure of either the local or constitutional derangement can be effected without skilful local treatment including strict observation of

every other local etiological factor. The author has noted many cases of serious pathological lesions in other parts of the body due to toxins supplied from oral sepsis treated by autogenous vaccines and imperfect local treatment, which resulted in little or no improvement, but when carefully treated locally by instrumentation and ionization were ultimately relieved of both local and constitutional affections and thereafter retained singularly effective immunity, which he is inclined to attribute in some degree to the effect of the vaccines. An instance of this may be recalled in a case of rheumatoid arthritis referred to on p. 51 which maintained recovery from the local and arthritic conditions in a striking manner after local treatment, but did not yield to a long course of vaccine treatments.

The local aspect of periodontal disease seems to be yet taken too lightly by the dental profession, who overlook or ignore the importance of incipient infection of the oral structures about the teeth, and do not realize that periodontal disease is preventable. As a rule the progress is slow and ample opportunity is presented to forestall the development of that stage which is termed pyorrhœa. The coöperation of the patient is more easily obtained in the early stages, when it does not impose such difficulties as arise later on. The habitual scrutiny of the gingival trough and removal of every particle of irritant from the teeth of all patients is not only of incalculable value to patients, but makes the operator expert in the instrumentation necessary when cases come under treatment suffering from advanced pathological changes, often due to perfunctory treatment or lack of any treatment, owing to pessimistic opinion prevalent on the prognosis of all cases of pyorrhœa.

CHAPTER VII.

PROGNOSIS OF PERIODONTAL DISEASES.

A glance at recent literature on the subject is sufficient to convince anyone that widely divergent opinions exist on the important point of the cure of pyorrhœa. It is not uncommon to find an article on the subject prefaced with the question: "Can pyorrhœa be cured?" or the assertion "pyorrhœa can be cured," or the conclusion at the summing up of an article "the disease cannot be cured."

The author has avoided the word "cure," while the principal endeavor has been to point out, not only that it can be cured, but that it should be the duty of every practitioner to approach the subject with that purpose in mind, on conservative lines. In the incipient stages of periodontal disease, it is generally conceded that the disease is curable, but the difference of opinion begins where the strict use of the word "pyorrhœa" is applicable. Those who have mastered the surgical technic and etiological problems in their method of treatment doubtless have ample clinical proof of the cure of pyorrhœa, knowing full well that the duration of that cure rests with the patient, and the gratifying results are due to coöperation of the patient; nevertheless, that their treatment and advice has effected a cure cannot be doubted. The object of every practitioner should be to effect a cure on the most conservative lines without undue risks in cases of pathological lesions in the systems being aggravated by recurrence

from whatever cause—(sometimes either negligence or inability on the part of the patient), bearing in mind that with elimination of oral sepsis the toxemic effect from that source will be relieved and the general improvement reflected at the local site of affection. Constitutional derangement is far too often attributed to oral infection, while that is only reflexly augmentary, and wholesale extraction of teeth advised by pessimists to the ultimate detriment of the condition of the patient who loses the vital asset of a functional set of teeth, often without improvement in the systemic lesion.

From carefully kept statistics the writer chronicles the following cases of cures:

I. Mrs. M., age about thirty-eight years, first seen on September 27, 1901. Periodontal disease general, suppuration about left lower incisor and right first premolar which were so loose that extraction of these two teeth was necessary: the superior left molar and premolar region also badly affected.

Treatment consisted in instrumentation, polishing and ionization with zinc ions, the disease was arrested after six treatments; the case has been seen twice or three times yearly, when only cleaning was necessary. There has been no recurrence of disease, on the contrary, in the regions of the worst affection the alveolar bone has developed abnormally. When last seen in March, 1919, the gums were perfectly normal. There seems to be no reason why these teeth, which are unusually strong, free from caries, and set in well developed jaws, should not continue in this healthy state as long as their owner lives, provided she is able to continue her method of daily hygiene, which includes the use of silk. This is indisputably a cure of pyorrhœa, even if in future the disease recurs it cannot affect the claim of its having been cured.

II. Mr. A., aged forty-two years, first seen October 11, 1910. Sent by Doctor with warning of existing syphilis: two mucous patches, general health badly impaired. Radiographs indicated considerable rarefying osteitis about lower incisors which were loose (Fig. 53), periodontal disease general. Etiological factor was subgingival calculus, revealed by *x*-rays. Local treatment consisted in instrumentation, polishing and ionization with zinc and iodine ions; eight treatments—suppuration ceased and gingivus healthy. Patient seen twice a year subsequently when cleaning was done and



FIG. 53.—Rarefying osteitis and enlarged sockets.

occasionally ionization of lower incisors which have not only remained healthy, as well as other parts of the mouth, but when last seen in January, 1919, the patient remarked that the once loosened teeth were firmer than he remembered them to have been for many years. Without treatment it is probable that every tooth of this denture would have been lost within these nine years, instead of being healthy and functional as they now are.

III. Miss R., aged about thirty years, first seen February 14, 1911. General health impaired, neuritis in left shoulder. General periodontal disease which was in advanced stage of

pyorrhœa in the maxillary incisor region due to malocclusion; deep pockets on palatal surfaces and teeth much displaced outward.

Treatment consisted in instrumentation and ionization with zinc ions, displaced superior incisors were retracted and inferior incisors ground clear of the bite; a retaining plate with gold wire arch was used constantly at first and always at night since; all suppuration ceased and the condition of the gingivus became normal after twelve treatments. Constitutional derangement disappeared in three months, and the patient was free from neuritis which had existed for two years. The patient, who lives in the country, has only been seen once a year. There has been no inflammation of the gingivus since and salivary calculus well kept away. When last seen in February, 1919, the general health was good, there had been no neuritis, and the teeth were firm with unmistakable clinical evidence of regeneration of bone about the incisors.

IV. Mrs. P., aged about fifty-five years. Seen April 29, 1910. Periodontal disease general, suppuration principally in the molar region on each side where well constructed bridges had supplied lost teeth for fifteen years. Rheumatic symptoms of increasing severity had appeared about a year previously.

Treatment consisted in removing the bridges and extracting all diseased roots; their places supplied with plates; instrumentation and ionization with zinc ions, nine treatments. The disease was completely arrested and the gingivus made healthy. The rheumatic symptoms were very much less in six weeks, and continued to improve during the following year. The case has been seen every six months and the gingival margin kept healthy by cleaning and occasional

ionization when symptoms of inflammation appeared at any place. Constitutional derangement has completely disappeared and any tendency to recurrence of the local lesion readily yields to treatment which the patient is glad to apply for.

These are but a few examples of what may be termed cures of pyorrhœa which probably any practitioner who endeavors to cure pyorrhœa at all can verify in his personal experi-

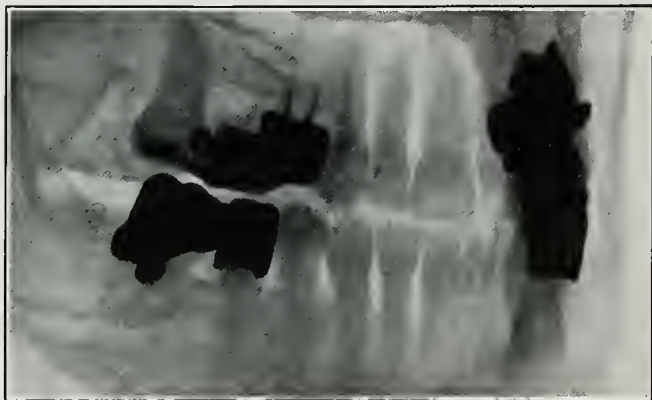


FIG. 54.—Radiograph of condition.

ence. Still there exists in this country a section of the profession who look on such cases as hopeless and attempt no treatment but extraction. Many there are who either mislead their patients by not mentioning the fact of existing periodontal disease until it cannot be longer kept from them when extraction is advised, or fail to diagnose it in the early stages and are pessimistic as to prognosis when pus supervenes.

The public undoubtedly is to blame for much of the pre-

vailing appalling oral sepsis now prevalent in this country which, but for nature's part in providing a barrier to the absorption of toxins, might well amount to a national calamity; if toxins from the mouth are accountable for as many ills as some medical men assert, there would be only a small proportion of really healthy people in the community.

CHAPTER VIII.

NOTES ON IONIC MEDICATION.

Definition—Ions—Electrolytes—Dissociation of Ions—Electro-positive and Electro-negative—Ions and Velocities of Ions—Depth of Penetration—Density—Therapeutic Effect of Various Ions—General Considerations.

Definition.—Ionic medication is that method of treatment in which electricity is employed to set in motion in a definite direction the soluble constituents of an electrolyte. The tissues of the body are conductors of current and ionic medication is the introduction of drugs into the tissues by conduction of the current, which modifies and changes the chemical constituents of the drugs employed and the tissues through which the current passes.

Ions.—The term “ions” was employed by Faraday to mean a moving particle in explanation of the phenomena observed when electrical currents are passed through electrolytes, and is employed electrically to imply the migration in a conducting solution of the chemical products of decomposition—, those which are positively charged, “kations,” move away from the positive poles, and those negatively charged, “anions,” move away from the negative pole. The direction of electrically charged ions is toward the opposite pole, and there is a double movement of ions going on, the kations toward the negative and the anions toward the positive pole. The ions of a salt differ from the atoms of that salt in that they convey electric charges, and are the conveyers of electric current in a solution.

Electrolytes.—Many chemical substances which are soluble in water are good conductors of electricity, and are called electrolytes. Pure water is a bad conductor, but the addition of a salt or acid increases the conductivity, so that although the water is a medium necessary in the composition of the electrolyte, it is the salt or the acid which conducts. The substances which in water form good conducting solutions undergo chemical decomposition when a current is passed through the solution, and the ions migrate in definite directions to either pole. The body is an electrolyte rich in ions, which, when a current is passed, are the means of conduction, and the quantities of its ions which migrate bear a direct proportion to the amount of electricity which passes, and the electro-chemical equivalent of its constituents.

Dissociation of Ions.—The theory of dissociation of a salt in a solution is explained by Arrhenius and provides an explanation of conduction of currents by ions in solutions. The solvent action of water on a salt splits up the molecules of the salt into ions of its chemical composition, so that a solution containing sodium chloride, for example, contains sodium chloride in part dissociated into Na ions positively charged and Cl ions negatively charged. In a solution through which no current is passing the ions move in no definite direction, but when a current is passed the movement of ions takes place in a definite direction, the positively charged Na ions toward the negative pole, and the negatively charged Cl ions toward the positive pole, and it is a double movement at the same time between the poles. The ions of metals, alkaloids, ammonia and hydrogen have positive charges, the ions of acids and hydroxyl (OH) have negative charges. Ocular demonstrations of the movement of ions

have been furnished by Leduc,¹ Lewis Jones,² Finze³ and the author⁴ has experimentally shown that ferrous ions migrate from the positive pole about an iron electrode immersed in egg albumen, in which ferricyanide of potassium has been incorporated; when a current is passed ferrous ions migrate away from the iron electrode toward the negative pole and can be seen moving in the electrolyte, which colors the ions blue. Abundant proof from a clinical standpoint has been chronicled by medical and dental writers of the migration of ions into the tissues by the action of the current.

Electro-positive and Electro-negative Ions and Velocities of Ions.—From the foregoing it has been pointed out that the action of the current on certain molecules in solution is to split them and repel the ions in a definite direction, the ions which are positively charged are repelled from the positive pole and carry a positive charge with them, these are electro-positive; those which are negatively charged are repelled from the negative pole, these are electro-negative. The basic radicals are positively charged, and the acid radicals are negatively charged. There are many substances which are frequently employed in electro-therapeutics, that are formed by the union of metallic radicals and acid radicals, such compounds as sodium chloride, zinc chloride and sulphate, copper sulphate, potassium sulphate, etc., which when acted upon by the current separate into electro-positive and electro-negative ions. The direction of movement of ions contained in a solution must be known in order to determine the proper pole with which to apply the medicine. For example, if zinc ions are required from zinc chloride and the negative pole be applied

¹ Electricity in Medicine, p. 197.

² Ionic Medication, Frontispiece and p. 8.

³ British Med. Jour., November 2, 1912.

⁴ Sturridge: Dental Electrotherapeutics, 1st edition, p. 214.

to the site of medication, chlorine will be liberated, but no movement of zinc ions. The following table of electro-positive and electro-negative substances gives the electro-chemical equivalents calculated to show the amount in milligrams liberated by one milliampère for one minute according to Lewis Jones and also the relative velocities of different ions as measured by Leduc for the human body.

ELECTRO-POSITIVE (KATIONS).

	Milligrams per Milliampère Minute.	Relative velocities, Leduc.
Ammonium	0.003	1.56
Cocain	0.012	0.59
Gold	0.04	1.22
Hydrogen	0.0006	0.88
Magnesium	0.007	0.5
Mercury	0.062	0.8
Potassium	0.024	1.0
Quinine	0.234	0.62
Radium	0.066	
Silver	0.06	0.5
Sodium	0.014	1.6
Zinc	0.02	0.6

ELECTRO-NEGATIVE (ANIONS).

Bromine	0.049	0.9
Chlorine	0.022	1.0
Hydroxyl	0.01	1.27
Iodine	0.078	1.16
Salicylic acid	0.085	
SO ₄	0.029	0.58

“To estimate the quantity of an ion which will be introduced into the body in a given time by a given current requires the consideration of two factors. One of them is the electro-chemical equivalent, and the other is the ionic velocity of the ion concerned. Only the electro-chemical equivalent need be considered if we wish to calculate the amount of a substance liberated at the poles of an electro-

lytic cell, but the ionic velocity is also important in calculating the amount of an ion introduced into the body from without. If the two ions concerned have equal velocity, then the amount of each introduced at the two electrodes would be one-half of the figures calculated for the time, the current and the electro-chemical equivalent; but if the two ions concerned have different velocities, then the 'share of transport' of the ion with greater velocity will be greater than half, and that of the other ion will be less than half, in the ratio of difference of their velocities."¹

Depth of Penetration of Ions.—The introduction of ions into soft, good conducting tissues such as periodontal and gingival tissue is a much easier matter than through the skin and in this respect ionic medication of oral tissues is much more effective than when the skin intervenes. To ascertain the depth of penetration of ions the author experimented on the gingival tissue of a dog. Incisions were made about the roots of several teeth to the depth of shallow pyorrhœa pockets, into which was introduced a metallic electrode wound with cotton-wool and saturated with 3 per cent. ferrous sulphate. A current of 5 ma. was passed in each pocket for three minutes, the operation performed in the same manner as for ionization in treatment of periodontal disease. Sections of the jaw were removed with tissues *in situ*, thoroughly washed, and placed in a 10 per cent. solution of potassium ferricyanide; this colored the ferrous ions prussian blue. It could then be seen that the ions had penetrated the soft tissues in every direction, and also the alveolar bone to a depth of 15 mm. The ionized gum and periodontal tissue was cut in sections, and mounted for examination

¹ Jones, Lewis: Ionic Medication, p. 20.

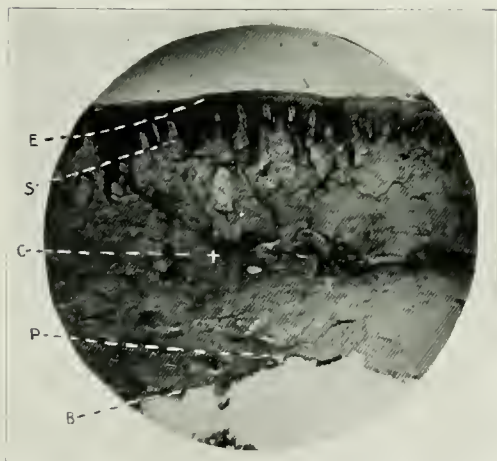


FIG. 55.—Vertical section of ionized gum. *E*, oral epithelium; *S*, papilla of submucous tissue; *C*, connective tissue; *P*, periodontal membrane; *B*, alveolar bone.

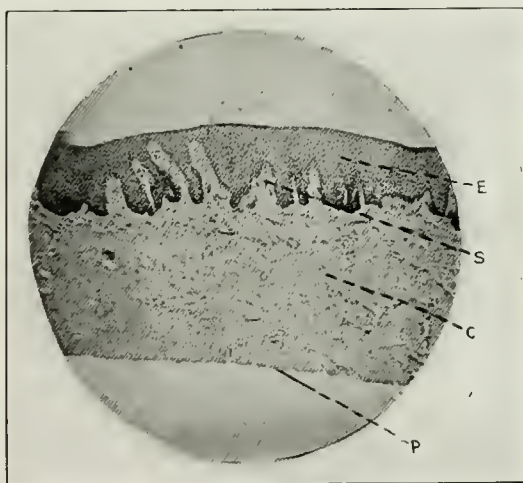


FIG. 56.—Vertical section of gum. *E*, oral epithelium; *S*, submucous tissue; *C*, connective tissue; *P*, periodontal membrane.

under the microscope. Sections were also prepared of similar tissue untreated. Fig. 55 shows photomicrograph of ionized tissue with a distinctly shaded appearance of the ions. Fig. 56 shows the contrast of untreated tissue.

The depth of penetration of heavy metallic ions is enhanced in the case of periodontal tissue, when application is made beneath the epithelial surface of gum tissue, and for that reason it is more effective to place the electrode into the gingival trough, rather than ionize through the gum surface.

Density.—The distribution of current on a charged conductor is on its surface; if the surface is spherical the distribution is all over the surface evenly, but if it is pointed it is greatest at the point, so also if it is knife-edged, the edges display the greatest density, and the flat surfaces are less charged. These facts have an important bearing on the method of ionic medication of periodontal tissue, inasmuch as the small spear-shaped electrode which it is possible to introduce into so small an area intensifies the current and furnishes considerable penetration of ions with but a very small current strength in a very short time. Conduction is more perfect, and penetration quicker and deeper, than in many applications on other parts of the body, where the epidermis has to be penetrated and the electrode is of larger area, even though larger currents may here be employed.

Therapeutic Effects.—The effects of different ions electrolytically introduced into tissues have been observed and chronicled by many writers, principally from a clinical standpoint, but experimental evidence has also been recorded to show that certain ions possess marked antiseptic properties. Under the heading of antiseptic metallic ions, zinc, silver, copper and mercury have been specially mentioned for their antiseptic qualities when introduced into tissues infected

with microorganisms. These ions are also well tolerated by the tissues and promote healing. Although recently some doubt has been thrown on the sterilizing effect of these ions, the author is convinced that there is no real cause to doubt their antiseptic and healing effect on septic periodontal tissue. Ample clinical evidence of unmistakable nature dispels every shadow of doubt, and places these metallic ions among the most effective in sterilization of oral tissues, and promoting a metabolic change in tissues weakened by disease.

Apparatus.—The source of current for ionic medication must be a continuous steady current, so controlled that only a fraction of a milliampère increase of current is permissible from the generator, when the resistance is released. A galvanic cell battery is a safe and convenient source of current which, if properly equipped with a reliable rheostat, answers the purpose admirably, but these batteries are often made for general medical purposes with current collectors, which increase and decrease the current abruptly. This kind is quite useless for the treatment of oral tissues. The battery should have a finely graded rheostat controlling the entire output of current and releasing it by a fraction of a milliampère at a time (Fig. 57). A milliampèremeter is essential and one with a large dial reading $\frac{1}{10}$ ma. to 5 ma. with a shunt in case larger reading is required, is the best.

Dynamo Current.—Continuous current from the main is the most satisfactory source of supply; the principal requirements in this case are a steady flow of current well controlled and so regulated and separated from the power-station dynamos that sudden rise or fall of current is impossible. The ionization apparatus of the Ritter Unit Equipment fulfills all the requirements for a safe and reliable outfit (Fig. 58).

The principle here is to generate current by a small noiseless motor which is separately wound and worked by either alternating or continuous main current which it transforms into low voltage and sufficient amperage for ionization.

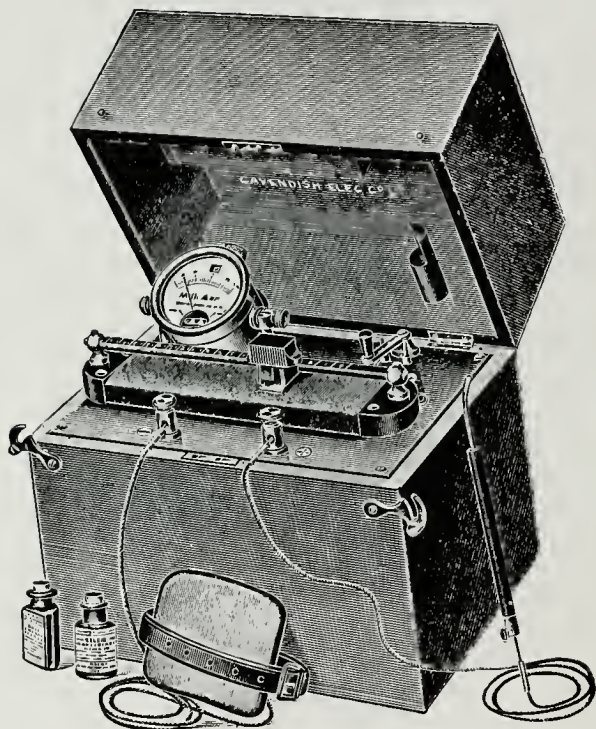


FIG. 57.—Galvanic battery.

One of the advantages of a motor transformer is the elimination of possibility of shock from short-circuiting with earth, which is a contingent to be guarded against with a main current switchboard.

A finely graded rheostat or current controller is the advantage of this particular outfit, the current can be so gradually increased that it is imperceptible until the limitation of the particular patient is reached.

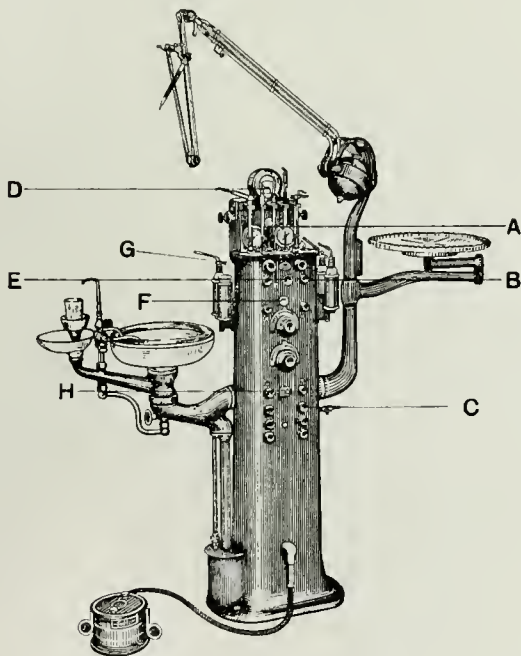


FIG. 58.—The Ritter unit equipment. *A*, pilot lamp; *B*, ionization generator switch; *C*, ionization generator fuse; *D*, milliammeter; *E*, ionization regulator button; *F*, ionization regulator dial; *G*, master switch; *H*, ionization terminal socket, patient circuit.

A large-dialed milliampèremeter with a scale of $\frac{1}{20}$ ma. registers the current strength in use.

The Ritter Company also supply a portable Ionization Outfit (Fig. 59) which is practically the same as the Unit

Equipment, suitable for those who do not require the larger outfit. This apparatus is admirably arranged for ionization work, possessing all the advantages of the transformer principle.

Switchboard for continuous current from the main is another means of controlling current for ionization (Fig. 60). In this

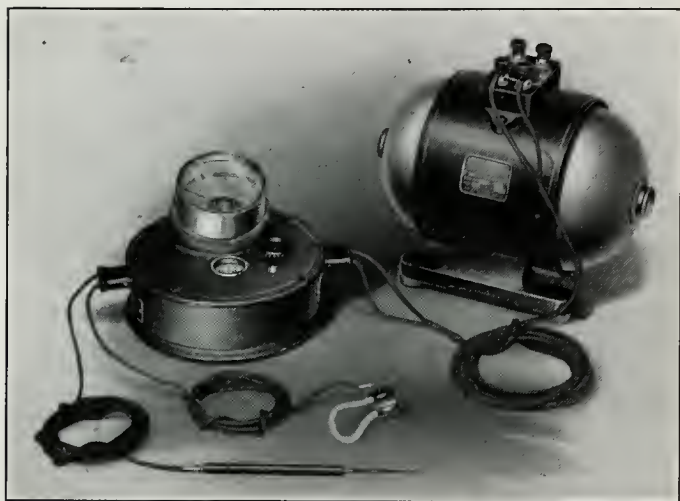


FIG. 59

case the chair should be insulated by a rubber mat under the base, metallic supports for cuspidor, water or gas pipes or any kind of metal in contact with the earth should be avoided, the saliva ejector must not be used. The current, after passing the rheostat on the switchboard, should be further controlled by a graphite rheostat (Fig. 61). This should be connected to the + terminal of the switchboard, and control the current passing to the patient, the indifferent electrode being

attached to the — terminal of the switchboard; the current is thus doubly controlled. This graphite rheostat has a resistance of about 20,000 ohms, so that by releasing about 20 volts pressure on the switchboard the current is completely

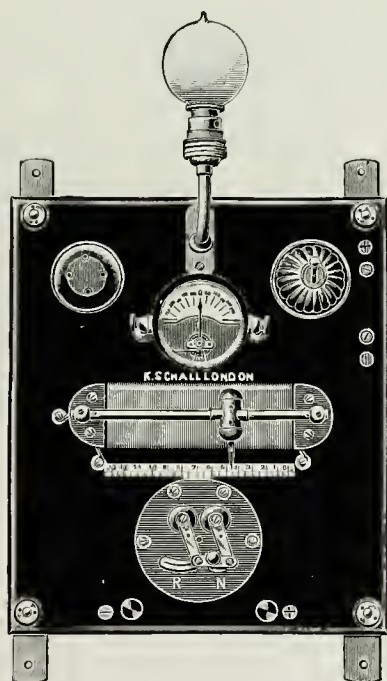


FIG. 60.—Switchboard for ionic medication.

resisted by the second rheostat, and by turning the glass dial it is released by a very gradual increase which is not perceived by the patient until sufficient current is obtained.

Conducting cords should be of best quality, and secured firmly both to the generator and electrode handpiece. Loose

contacts cause rise and fall of current which is disagreeable to the patient.

The poles should be tested after all connections are made by placing the two electrodes intended for use on a patient a

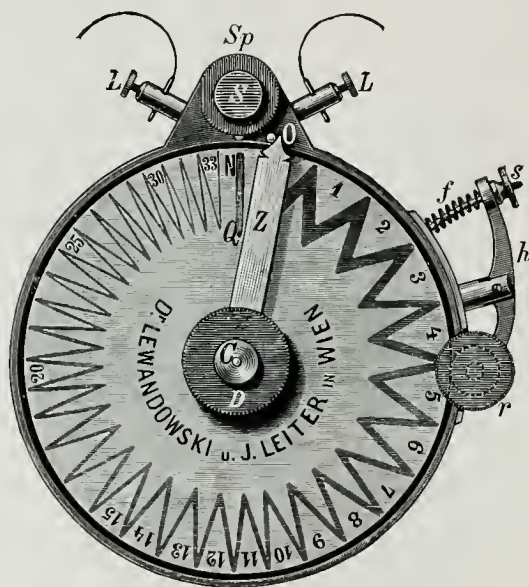


FIG. 61.—Graphite dial rheostat.

short distance apart in a glass of water to which has been added a few drops of phenolphthalein. On passing a few milliampères of current a purple coloring of the water will take place about the negative pole. When the correct poles are found (according to the + and - signs on the switchboard) the deflection of the milliamperemeter needle should be noted, if it deflects to the left of the dial, it must always deflect in that direction in future use to correctly correspond to the

marking of poles on the switchboard. A mark should be made on the wall plug and switchboard plug to ensure its being connected in correct polarity in future.



FIG. 62.—The author's pyorrhœa electrodes.

Electrodes for ionization of periodontal tissues should be of zinc, copper, or platinum, shaped to pass readily into gingival trough, interspaces or deep pockets (Fig. 62), and as large as possible compatible with the areas into which they must pass during treatment. Platinum can be used with any solution, but zinc and copper should only be used with solutions corresponding with those metals, like zinc chloride or copper sulphate.



FIG. 63.—The author's electrode handpiece.

The electrodes should be interchangeable so that they can be readily removed for sterilization, and attached firmly by a clutch at the end of an insulated handpiece.

The indifferent electrode should be of metal (aluminum or nickel-plated brass) or carbon, and should be covered with

several layers of lint, forming a pad between it and the skin at the point of contact. The area should be large but the

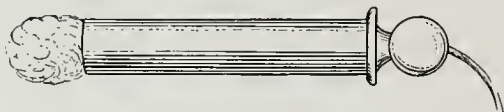


FIG. 64.—Hand electrode.

site of contact does not materially matter. The hand electrode (Fig. 64) should be firmly held by the patient.



FIG. 65.—Wrist electrode.

The wrist electrode (Fig. 65) should be tightly strapped into position, so also the chin electrode.

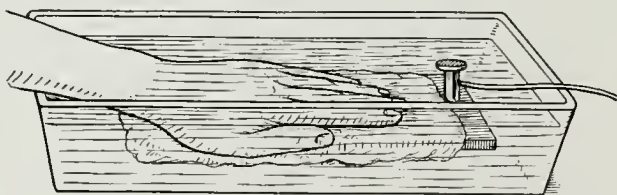


FIG. 66.—Carbon and water electrode.

In case the metallic electrode causes blisters on the skin or is painful, a carbon and water electrode can be used with comfort, a little sodium chloride placed in the dish of tepid water and a rubber-covered conducting cord completes this form of contact.

General Considerations.—There are many minor points which arise during treatment of oral tissues, which tend to frustrate the use of electricity in these parts. These can always be overcome when full knowledge of the phenomena which govern these causes is possessed by the operator, who should be able to inspire the patient's confidence by his command of the work. Variations in resistance of the tissues at the site of contact; the difference in sensibility of root surfaces of different teeth and the presence of metallic fillings in sensitive cavities adjacent to parts being treated, are the most frequent causes of discomfort. These should be taken into consideration during the seance. If the tissues are thin and wasted or the gingival trough shallow, it is unnecessary to ionize it as long or with so great a current, as when they are thicker and deep pockets exist. So, too, if roots of teeth exhibit sensibility to current in these areas a small current will be sufficient where the greatest sensitiveness is present, as is often the case about the incisors. An excess of solution on the well covered electrode and keeping the electrode steady will permit of a greater current at those points, care being taken to reduce the current before moving on the metal point to an adjoining part. When metallic fillings are the cause of discomfort, the same precaution of steady contact will often overcome this difficulty, and care should here be taken not to abruptly move the electrode, suddenly breaking its contact with the filling, as this might stimulate the nerve filaments in connection with the nerves of the eye, and cause the impression of a flash of light, which is somewhat alarming to the patient.

The current should be turned on gradually and the limitation of the patient's endurance tested up to the point, not only that it is being slightly felt, but in order that sufficient

current is obtainable to secure proper penetration of ions, and also not to prolong the time. The endurance of slight discomfort should be requested during which it will often be found possible to raise the current strength considerably without increase of discomfort or pain. The best results are always obtained for patients with the least resistance to current, and those who are able to take 5 to 15 ma. with comfort. It should be the object of the operator to obtain at least 5 ma. in the molar regions and 3 ma. in the incisor regions.

The application should be made directly into the gingival trough with an electrode capable of entering it, and the solution conveyed on cotton-wool, which should be wound about the point of metal. No reliance can be placed on time-saving electrodes, such as can be found on the market, which are intended to ionize the entire gingival border at one time with the hope of penetrating the gum tissue and sterilizing the underlying gingival trough or pyorrhœa pockets. Such applications are not direct enough, and the penetration of ions is too superficial with the current strength which oral tissues will tolerate.

For further information on electro-physics and electro-therapeutics applicable to dental use the reader is referred to larger medical and dental works on the subject. The author's work, *Dental Electro-therapeutics*, has been compiled with the object of collecting and condensing this information for dental use.

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